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DR. MAX A. GOLDSTEIN

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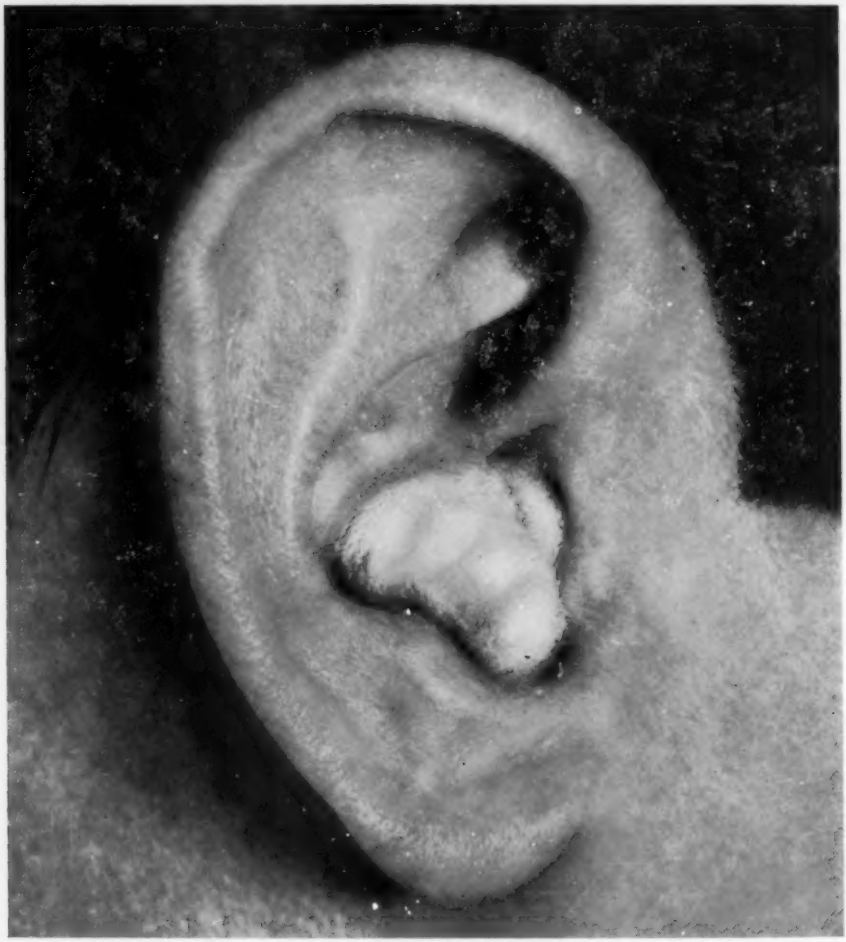

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THE
LARYNGOSCOPE.

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SIXTY-FIVE YEARS IN OTOLARYNGOLOGY.

Just 65 years ago, in July, 1896, the first number of THE LARYNGOSCOPE was published. The founders were Drs. Max A. Goldstein and Frank M. Rumbold who, on their return from graduate study in Europe, realized the need for a monthly journal devoted to the diseases of the ear, nose and throat. In the first number they announced that the journal would be for "General Practitioners and Specialists" and hoped that it would "interest that large body of progressive physicians to be found in every town and city, who, from time to time, are called upon to treat diseases of the nose, throat and ears." They chose the name LARYNGOSCOPE for their journal because they thought it would "broadly designate an illuminating instrument for use in the examination of diseases of the nose, throat and ear."

The original articles were definitely of a practical nature and much of the journal was devoted to abstracts of practical articles from foreign literature. At the outbreak of the Spanish-American War, Dr. Rumbold, who had been very active in the National Guard, enlisted in the Army, and in Volume II of 1899 his farewell editorial appears. Dr. Goldstein carried on as editor until his death in 1941. In February 1942 Dr. Theodore E. Walsh became the editor.

In the earlier issues a large part of the journal consisted of reports from the literature of other countries. Nowadays the original articles occupy almost the complete journal. Another interesting change is in the price. The original LARYNGOSCOPE was published at the subscription price of $2.00 per

annum. Would that the dollar bought as much today as it did then!

We hope that with the continued support of our editorial board, our contributors, our subscribers and our advertisers, THE LARYNGOSCOPE will serve otolaryngology as well in the future as in the past 65 years.

ANNUAL OTOLARYNGOLOGIC ASSEMBLY.

September 23 through 30, 1961.

The University of Illinois College of Medicine Department of Otolaryngology will offer an intensive postgraduate basic and clinical program under the direction of Doctor Emanuel M. Skolnik. This Assembly for practicing otolaryngologists offers a compact program of one week of daytime and evening sessions. It is designed to bring to specialists a wide variety of current advances in management, therapy and philosophies. Review of basic morphologic features under the direction of Doctor Maurice F. Snitman and Doctor Frederic J. Pollock is also included, and will feature laboratory demonstrations, dissection and prosection, all augmented by visual aids.

Panel programs have been designed to bring out special features of otologic and reconstructive surgery and tumors of the head and neck. Luncheon chats are an important part of the daily program.

Interested physicians should write direct to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

PHYSIOLOGY AND PATHOLOGY OF THE CRICOTHYROID MUSCLE.*†‡§

GODFREY E. ARNOLD, M.D.,
New York City.

INTRODUCTION.

Although the cricothyroid is one of the smallest muscles in the human body, it has received a great deal of attention in basic and clinical research. For a long time much information has been available on the details of its anatomy, function, and significance for vocal performance, yet, this information has not gained entrance into clinical practice for the usual reasons: the data are scattered in specialized journals, some in old or rarely available volumes, and many studies were written in languages other than English. As a result, several recent authors reported on some clinical aspect of disturbed cricothyroid function without being aware of the previous work. Certain misconceptions have also crept into clinical thinking, and the laryngologic practitioner is often confused when confronted with the possibility of cricothyroid paralysis which occurs much more frequently than is recognized.

The purpose of this paper is threefold: one, to assemble the scattered data and generally accepted facts concerning cricothyroid function; two, to clarify certain problematic details of vocal cord tension by personal experimental contributions; three, to describe the characteristic clinical syndrome of neurogenic or myopathic cricothyroid paralysis as it emerged from isolated previous observations and systematic personal studies.

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†From the Department of Research, New York Eye and Ear Infirmary.

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The findings will be presented according to the *method* of investigations used by previous authors and in this report: *1.* Anatomical considerations, *2.* Nerve supply, *3.* Muscular action, *4.* Electrophysiologic experiments, *5.* Vocal physiology, *6.* Lateral radiography, *7.* Frontal tomography, *8.* Electromyography, *9.* Laryngoscopic findings, *10.* Phoniatric voice analysis, *11.* Diagnostic procedures, and *12.* Therapy. This integration of new findings with accepted facts should lead to a clear portrayal of this characteristic but little known paralytic syndrome for the benefit of prompt clinical recognition.

TABLE I.

Mean Values of Size of Vocal Cord Tensors.
(After Faaborg-Andersen.)

Muscle	Length in mm.	Breadth in mm.	Thickness in mm.	Cross section in mm.^{2}	Volume in mm.^{3}	Calculated weight in mg.	Actual weight in mg.
Cricothyroid	15	10	6	60	900	954	940
Vocalis	15	4	5	20	300	318	357

ANATOMICAL CONSIDERATIONS.

1. The *morphology* of the cricothyroid muscle has been well known for a long time and need not be described again. Historical details of the development of anatomical knowledge were described by Tschiassny.[70] It is generally accepted that the muscle consists of two main portions: the *pars recta* and the *pars obliqua*. Some authors[53,70] described a third portion, the *pars interna*, which extends from the upper cricoid edge to the inner plate of the thyroid. Available data[11] on the dimensions of this small muscle are shown in Table I.

According to Pernkopf's[60] detailed description, the muscle *originates* along the inferior margin of the thyroid wing. The ventral *pars recta* extends from the midline to the inferior thyroid tubercle, and the dorsal *pars obliqua* from this tubercle to the inferior thyroid horn. The third or internal portion[53] originates from the inner plate of the thyroid wing and its

inferior horn. The *insertion* is seen on the ventral and lateral aspect of the cricoid ring. Other authors[53,70] reverse the description of origin and insertion. It is elementary knowledge that this course determines the muscle's main activity: to approach the anterior parts of the thyroid and cricoid cartilages.

2. Cricothyroid joints: Anterior approximation of the cricoid and thyroid cartilages is made possible by the two cricothyroid joints. These are situated between the articular surfaces on each side of the cricoid ring and the rounded tips of the two inferior thyroid horns. In most cases, the joints represent diarthroses; synarthroses in the form of syndesmoses or synchondroses are but rarely found.[78]

3. Cricothyroid ligaments: Exact details of the three ligaments around either joint, the Lig. cerato-cricoideum posterius, laterale, and anterius, were recently described by Mayet and Muendnich.[53] On the basis of their minute dissections, they accept three types of *articular movements:* mainly a rotation around a common frontal axis through both joints, and slight sliding movements in vertical and sagittal direction as long as the joints are in neutral position. When the crico-thyroid distance is either distended or contracted, the sliding movements are prevented by the articular ligaments because they are then in a state of tension.

4. Average values for the *crico-thyroid distance* as a direct measurement of anterior crico-thyroid approximation were indicated as follows: 20 mm. for extreme distention, 12 mm. in neutral rest position, and 3-6 mm. for extreme approximation.[53] It should be borne in mind that such absolute averages found on *anatomic preparations* vary considerably with sex, age, laryngeal dimensions, voice type, etc.; moreover, they differ from the relative values obtained by measuring the *radioscopic projection* of the crico-thyroid distance and its functional changes in the living. These radioscopic values will be discussed later.

NERVE SUPPLY.

1. Laryngeal nerves: Many authors[33,34,70] concede that

Longet,[41] in 1841, was the first to describe the innervation of the cricothyroid muscle by the *superior laryngeal nerve* (N. laryngicus cranialis). In spite of considerable controversy around the complicated and variable details of laryngeal innervation,[5] it is now absolutely established that the *external* branch of the superior laryngeal nerve is the motor nerve of the cricothyroid muscle, whereas the *internal* branch of this nerve provides the sensory innervation of the upper larynx to the level of the glottis.[27,32-34,41,70] Below this level, the recurrent or inferior laryngeal nerve (N. laryngicus caudalis) transmits the sensorial supply of the lower larynx.[51] It seems definitely established that the internal branch of the superior laryngeal has no motor function.[52,56]

2. The problem of a *double innervation* of certain laryngeal muscles by both laryngeal nerves fascinated the older authors. Relying mostly on animal experiments, they often came to divergent conclusions. Katzenstein[33] discussed the question of a double innervation of the cricothyroid by *a.* the superior laryngeal nerve and *b.* Exner's N. laryngeus medius. He concluded[24] that Exner's nerve exists only in rabbits, constituting a separate branch of the ramus pharyngeus nervi vagi. Stupka[67] confirmed the existence of this nerve also in the dog's larynx. Its stimulation produced contraction of the oblique portion of the cricothyroid. No such nerve is recognized in man, possibly because the main mechanism of human vocal cord tension is mediated by the *pars recta.*

3. The *segmental innervation* of the larynx, both motorically and sensorially, by the upper and lower laryngeal nerves, is explained by embryologic considerations. According to Pernkopf,[60] the laryngeal muscles develop from the two pharyngeal sphincters—the external and the internal.

The *external* pharyngeal sphincter has again two derivatives: a dorsal and a ventral. *a.* Its dorsal derivative becomes the laryngopharyngeus muscle with its two portions, the thyro- and cricopharyngeus; *b.* its ventral derivative is the cricothyroid muscle. Originating from the fourth branchial arch, this muscle is innervated by the *fourth branchial nerve,* which is the superior laryngeal nerve, via its external motor

branch. It appears quite logical that the internal sensory branch of this nerve should transmit all afferent sensations from the upper larynx, which has the same branchial origin.

Derivatives of the *internal* sphincter are the intrinsic laryngeal muscles. Being derived from the sixth branchial arch, all internal laryngeal muscles are motorically supplied by the *sixth branchial nerve*, namely the inferior (recurrent) laryngeal nerve. Their proprioceptive sensation is likewise transmitted through the sensory fibers of the recurrent nerve, as are the afferent sensations from the infra-glottic lower larynx.

MUSCULAR ACTION.

1. Cricoid Movements.

When the cricothyroid muscle contracts, it approximates the anterior portions of the cricoid and thyroid cartilages. It is curious that many authors were confused about the simple question which is the moving part. Tschiassny[70] stated that Magendie, in 1813, was the first to describe the cricoid as the moving part. In many subsequent contributions this action was falsely ascribed to the thyroid. Since two cartilages are involved, about 50 per cent of the authors had the wrong cartilage in mind when describing its movements; therefore, it was felt necessary to explain the true relationship over and over again.[58]

Because the thyroid is fixed by its external strap muscles to the hyoid and sternum, it cannot be moved by the small cricothyroid muscle. The cricoid, on the other hand, has no muscular connection with the skeleton and can freely elevate the mobile trachea; hence, the *cricoid moves* in response to cricothyroid contraction. This view is now shared by most authors.[2,11,15,20,24,29,30,38,52,56,58,60,63,70,75]

2. Modes of Vocal Cord Tension.

Since it is established that the cricoid cartilage moves with contraction of the cricothyroid muscle, anterior elevation of the cricoid ring narrows the cricothyroid distance. This is the visible and palpable sign of cricothyroid muscle action.

As a result, the posterior cricoid plate tilts backward and downward. Carrying the arytenoids by means of the cricoarytenoid joints, this dorsad movement of the cricoid plate elongates the distance between the anterior thyroid insertion of the cords and their posterior insertion on the vocal processes. Consequently, the *vocal cords are stretched* by cricothyroid contraction. These mechanisms are beautifully illustrated in Pernkopf's anatomy.[60]

It follows that the cricothyroid's primary function is that of crude or *external vocal cord tension.* Because this tension is achieved by increasing elongation of the cords with rising pitch, it is an isotonic tension. In contrast, the fine or *internal cord tension* from subsequent contraction of the internal thyroarytenoid (or vocalis) muscle occurs with equal cord length for a given pitch level, and represents an isometric tension; consequently, the cricothyroid and the vocalis are synergists with different modes and purposes of function.

Studying the complicated system of vocal cord tension, Zenker and Zenker[73] assume the following muscular interactions: *a.* The vocal cords are *elongated* by the cricothyroid, assisted by the thyropharyngeus which compresses the thyroid wings and thus elongates the sagittal diameter of the larynx; *b.* The vocal cords are *shortened* by the cricopharyngeus which tilts the cricoid plate upward and forward; in addition to this antagonism between cricothyroid and cricopharyngeus, the tracheal pull adds to the shortening effect by lowering the cricoid ring, especially when the larynx is elevated with high tones. This antagonism was confirmed by EMG findings, to be discussed later.

3. Auxiliary Adduction.

In view of the separate portions of the cricothyroid muscle, their contractions can be analyzed further. As Mayet and Muendnich[53] have shown, the vertical *pars recta* elevates the cricoid ring; the more horizontal fibers of the *pars obliqua* cause a horizontal dorsad retraction of the entire cricoid in relation to the thyroid; and the *pars interna* is thought to have a constricting action on the glottis.

Since the days of the old controversy between the adherents of Wagner and Grossmann's theory of the paramedian position of a completely paralyzed cord and the followers of Rosenbach and Semon's rule of intermediate position in complete recurrent nerve paralysis, the question of the cricothyroid's adducting function has intrigued many observers. It is now believed that the external tensor *contributes to vocal cord adduction.*[5-7,16-18,27,53,58,62]

Discussing the adductor function of the cricothyroid, Tschiassny[71] agrees with the modern concept that in pure *recurrent nerve paralysis the cord is in paramedian position,* held there by this muscle.[5,16-18,62] The constant adduction to paramedian position is maintained by reflex contracture of the cricothyroid. This author[71] recommended three tests to ascertain the influence of cricothyroid function in cases of bilateral vocal cord paralysis in paramedian position.

a. Cocainization of the larynx to interrupt the afferent limb of the reflex arc: when this is followed by widening of the glottis, the cricothyroid should be eliminated to interrupt the efferent limb of the reflex contracture. *b. Probe test of arytenoid* to determine its passive mobility: when it is found immobilized, arthritic ankylosis is the cause of paramedian fixation. *c. Probatory cricothyroid elimination* by novocaine injection into the muscle: when this test is followed by widening of the glottis, an absolute indication for the ablation of this muscle is present.

The possibility of widening the glottis in bilateral midline paralysis by elimination of the adducting cricothyroid has interested many authors for a long time.[3,5,7,71] After it had been confirmed in man[23] that section of the *recurrent* nerve produces *paramedian* position of the paralyzed cord, and that subsequent section of the *superior laryngeal* nerve (or ablation of the cricothyroid[3]) causes the paralyzed cord to shift to *intermediate* position, several authors recently re-affirmed this regular observation.[16-18,71]

For these reasons, Jeschek[28] recommended excision of the homolateral cricothyroid muscle in addition to arytenoidec-

tomy. Following such *cricothyroidectomy*, the glottis was shifted posteriorly to the side of excision, the previously narrow cricothyroid distance was much wider, and the homolateral cord appeared relaxed and in intermediate position. Reasons for occasional failures of this operation, such as ankylotic arytenoid fixation, were discussed by previous authors.[6,7,9,27,71]

4. Integration of Muscular Interactions.

To summarize, it can be stated that the cricothyroid muscle has a threefold function: *a.* It produces *tension of the vocal cords* by anterior elevation of the cricoid ring and lowering of the cricoid plate posteriorly[56]; *b.* It causes *auxiliary adduction* of the cords from the intermediate to the paramedian position[27,53] because tension of the cords rotates the vocal processes inward; *c.* It assists in *auxiliary abduction* from the median to the paramedian line, because both the vocal cords and the cricoarytenoid ligaments are anchored in the paramedian line.[53]

Jeschek[26] seems to be right in stressing the constant and *complicated interaction* between all laryngeal muscles. Apart from the *main* function of each muscle, there is an *auxiliary* function which assists the action of other muscles under certain conditions and within certain limits. The details of such auxiliary action depend on vocal cord position. As just discussed, the cricothyroid's auxiliary function includes both adduction and abduction, tending to keep the cords in the paramedian line.

Coordinated interaction of the extrinsic and intrinsic laryngeal muscles is important for *a. fixation* of the arytenoids and thereby of the cords, *b. crude vocal cord tension, c.* and ultimately for the *fine inner adjustment* of the cords according to each type and degree of phonic performance.

ELECTROPHYSIOLOGIC EXPERIMENTS.

Much information on laryngeal physiology was accumulated by the older authors who tried to recognize the main rules

of laryngeal innervation by animal experiments.[1,22-24] Providing the manifold differences between the human larynx and that of various animals are duly considered,[5] certain basic phenomena can be reliably established.[59,62,67]

1. Electrical Stimulation.

Stupka[67] elaborated numerous details of laryngeal innervation which supplemented and confirmed many of Neumayer's[59] previous findings. With his experiments on dogs he obtained the following results of *unilateral stimulation* of the cricothyroid muscle: *a.* Homolateral elevation of cricoid ring; *b.* Rotation of anterior cricoid ring towards the side of contraction so that the posterior commissure deviates to the other (non-active) side; *c.* Homolateral elongation and elevation of the vocal cord; *d.* Homolateral retraction of the arytenoid.

It should be obvious that unilateral elevation is due mainly to contraction of the *pars recta*, whereas the torsion of the cricoid ring in the horizontal plane results primarily from the backward pull of the *pars obliqua*.[70] While repeating these experiments with bilateral stimulation of the *cricothyroid* muscle, Muendnich[56] confirmed the impressive dorsad and caudad tilting of the cricoid plate. This posterior movement is much greater than the concomitant elevation of the anterior cricoid ring. What results is a strong tension of the cords with a firm and almost sphincter-like glottal closure.

When the *internal* branch of the superior laryngeal nerve was stimulated, a reflexive closure of the glottis resulted. After suppression of these reflexes by deep anesthesia, no such reflexive closure was seen. Again, it became obvious that the internal branch of the superior laryngeal nerve serves no motor function.[23,56,67]

Numerous experiments with unilateral stimulation of the entire *superior laryngeal nerve* above its division, showed blanching, tensing, elongation, and adduction of the cord on the stimulated side.[16,20,62] The typical torsion or rotation of the anterior cricoid ring to the side of stimulation was always confirmed.[20] *Bilateral* stimulation of the superior laryngeal

nerves produces elevation of the anterior cricoid ring, descent of the posterior cricoid plate, and visible tension of the cords.[20]

Stimulation of the superior laryngeal nerve, as part of a systematic study of the *neural influences on phonation*, was done by Isshiki.[25] With meticulous experimentation and registration he could show again that stimulation of this nerve leads to cricothyroid contraction which lengthens the cords and increases vocal pitch. Thus, cricothyroid contraction and the resulting vocal cord elongation is the most important factor for the increase of vocal pitch.

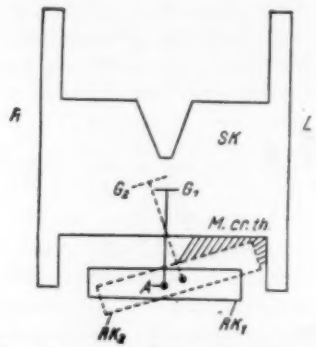

Fig. 1. **Unilateral action of cricothyroid muscle,** pars recta, anterior view. SK thyroid; RK_1 cricoid at rest; RK_2 cricoid with contraction of left cricothyroid. G_1 glottis level at rest; G_2 glottis level with contraction of left cricothyroid. A: sagittal axis of cricoid. M.cr.th.: left cricothyroid. R: right, L: left side of larynx. With stimulation of left cricothyroid, or with paralysis of the right muscle, the left half of the cricoid is elevated and rotated to left; this lowers the right vocal cord.

2. Interpretation of Cricoid Movements.

The significance of these complicated movements for the interpretation of unilateral cricothyroid paralysis is best explained by two diagrams. All one has to bear in mind is that paralysis produces the precise opposite result of electric stimulation. As Figure 1 shows, unilateral paralysis on the right side, or isolated stimulation on the left side, causes elevation of the cricoid ring on the left side, obviously from contraction of the *pars recta* on the active left side. As a result the affected, or non-stimulated right *cord will be lower* in

perfect agreement with experimental findings[67] and clinical observations.[2]

Figure 2 explains the action of the *pars obliqua* with either paralysis or stimulation of one side. Whenever one muscle contracts alone, it will rotate the anterior aspect of the cricoid ring to the side of contraction; consequently, the posterior glottal commissure will rotate to the opposite side. This means that in case of unilateral paralysis the *posterior commissure is deviated to the side of paralysis.*

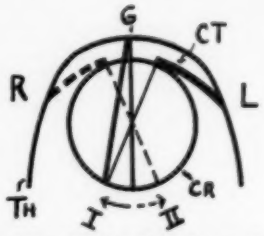

Fig. 2. **Unilateral action of cricothyroid muscle,** pars obliqua, laryngoscopic view. Th thyroid, Cr cricoid, G glottis axis. CT: left cricothyroid muscle. R: right, L: left side of laryngoscopic image.

I. **Stimulation** of left cricothyroid muscle rotates the cricoid ring to left and the posterior commissure to right. Note deviation of glottis to right side.

II. **Stimulation** of right cricothyroid muscle rotates the cricoid ring to right and the posterior commissure to left. (Deviated glottis not shown.)

I. With **paralysis** of right cricothyroid, the cricoid ring is rotated to left and the posterior commissure to right. Note deviation of glottis to right side.

II. With **paralysis** of left cricothyroid, the cricoid ring is rotated to right and the posterior commissure to left. (Deviated glottis not shown.)

Once these two components of *unilateral elevation and torsion* of the cricoid ring, as a result of unilateral cricothyroid action, are understood, it is quite easy to interpret properly the clinical signs of unilateral cricothyroid paralysis. Various authors are in agreement with these interpretations[9,16-18,20,21,28,53,72]; therefore, it can be stated that a cardinal sign of unilateral cricothyroid (or superior laryngeal nerve) paralysis is the finding of an *oblique glottis.*

There are only rare exceptions to this torque effect by unilateral action of the intact cricothyroid's oblique portion. These are pressure effects from an enlarged thyroid gland

which may also cause a deviation of the glottis, or of the entire larynx, to one side. In such a case, however, there will likely be present the additional finding of a compressed trachea which, of course, is absent in a case of pure cricothyroid paralysis.

BASIC CONCEPTS OF VOCAL PHYSIOLOGY.

1. Interdisciplinary Aspects of Voice Science.

Although the physiology of the human voice is known to a considerable extent,[2,4,9,29,31,49,60-63,69] some eccentric opinions delayed the general understanding of basic facts by the laryngologic profession. In view of the great amount of available information, it is surprising that as late as 1931 two authors[64,73] claimed earnestly that contraction of the cricothyroid muscles lowers vocal pitch. This false notion resulted from erroneous interpretations of improperly performed experiments.

Ever since Manuel Garcia, singing teacher and inventor of laryngoscopy, initiated the correlation of pedagogic empiricism with exact physiologic experimentation, phonologic research accepted the *vocal terminology* traditionally used in musical circles. Greatly aided by musical acoustics, the scientific study of the human voice has elaborated a good deal of knowledge which is widely accepted among students of the acoustic, physiologic, and pedagogic aspects of vocal communication by speaking and singing. Let us review briefly the *basic facts of vocal physiology* insofar as they are pertinent to the problem of cricothyroid function.

2. Vocal Range and Voice Type.

It is an elementary fact that the dimensions of the larynx, particularly of the vocal cords, determine the *vocal range* of each individual. In general, a large larynx with long and broad cords produces a range of low tones, whereas a small larynx with short and slim cords is tuned for a range of much higher tones. This fact represents one of the secondary sex characteristics: the male larynx is considerably larger

than the female larynx, and the adult larynx is larger than the child's; moreover, there are great individual differences.

These laryngeal dimensions are the main determinants of the six *human voice types:* bass, baritone, and tenor in the adult male; contralto, mezzo-soprano, and soprano in the adult female and the prepubertal child. Further *subtypes* of dramatic, lyric, buffo, or coloratura techniques that can be developed by each type depend on additional factors, such as body type, dimensions of supraglottic resonators, artistic inclination, vocal education, psychologic personality structure, cultural influences, etc. The three male voice types with their subtypes are one octave lower than the corresponding three female voice types and subtypes. This fact is clearly illustrated by the vocal data of subjects I and II.

As a general rule, the individual range of each voice type encompasses at least two octaves. Good singers possess greater ranges of three octaves or more. It is customary to distinguish the smaller *musical range* of artistically usable tones from the larger *physiologic range* which encompasses all tones that can be produced with effort. The greater the difference between the two ranges, the more reserve of pitch variation is available to a gifted singer.

Absolute frequency values of these ranges can easily be recognized either by the simple technique of pitch matching with tuning forks, audiometer, pitch whistles, and piano, or by oscilloscopic, sonagraphic, and other electronic recording (see Figs. 3 and 4). When all vocal ranges from the lowest bass to the highest coloratura are added together, one obtains the *collective human range* which spreads over 5 to 5½ octaves from about 50 to 2,000 cps.

It should not be forgotten that these frequencies refer to the *fundamental* tone, or first partial tone (harmonic), of laryngeal phonation. They have nothing to do with the higher harmonics which constitute the *formants* of oral speech sounds; the phonetic speech range of formants extends from 200 to 8,000 cps; clinical parlance refers to its practically important center from 500 to 2,000 cps.

3. Vocal Registers.

The vocal range of each voice type includes three *vocal registers:* chest, middle, and head. Their relative extension contributes to the acoustic characteristics of the vocal type. The low chest register is larger in the low types, whereas the high head register is more developed by the high vocal types.

At the borders of two adjacent registers, the *transitional* tones can be produced by the mechanisms of either register. This *passage,* or bridge, of registers is most noticeable between the male middle and head register. In the untrained natural voice, or in the phonasthenically weakened (dysodic) singing voice, an unpleasant *break* separates these two registers (see Figs. 15 and 16). The vocal technique of "covering" is used for the equalization of these register passages. It is still a puzzling mystery why the continuity of tones in the male voice should be interrupted by such a passage or break.

In addition to these *physiologic* main registers, the practice of singing recognizes several *artistic subdivisions: a.* The church bass register below the chest register of very deep bass voices (basso profondo).[49] *b.* The male head voice appears in three forms, namely *1.* the thin sounding natural falsetto (German: Fistelstimme, French: fausset) of the untrained voice, *2.* the delicate artistic falsetto (German: Falsett, false soprano) as a special decrescendo (fading) effect, and *3.* the loud full tone of the trained head voice with added resonance (French: voix mixte).[47,48] *c.* In the female, with only one type of head voice, there may be a flageolet (or whistle) register above the highest head tones of the coloratura soprano.[19]

4. Acoustic Definitions of the Examined Voice.

As an illustration of the foregoing statements and for the precise definition of the examined vocal qualities, the acoustic dimensions of the principal test person's voice will now be described. With one exception, all investigations were conducted with the same subject which permits direct comparisons of the various findings made on this person. Musical notation of the test tones is based on Helmholtz's classifica-

tion. Frequencies in cps are indicated according to the modern standard of concert $a^1 = 440$ cps.[49]

Subject I: Normal healthy male, age 45, height 6 feet, weight 165 pounds, with an untrained baritone voice, but good musical experience. Vocal range: *D* (73 cps.) to d° (147 cps.) in chest voice, d° to d^1 (294 cps.) in middle voice, d^1 to a^1 (440 cps.) in untrained falsetto voice. A gradual register passage at d°, and an abrupt break at d^1 marks his transitions. Average speaking range: *F* (87 cps.) to c° (131 cps.).

Recording of test tones. The specific vocal functions to be described later with several techniques were first acoustically recorded on the sonagraph. Gratitude for valuable assistance is expressed to Mr. H. L. Barney of Bell Telephone Laboratories, Murray Hill, New Jersey. The subject recorded the phoneme "Ha" (as in "father") on the tones *G* (98 cps.), d° (147 cps.), a° (220 cps.), and f^1 (350 cps.). In this manner, his tonal range and registers were well represented. The same tones and the same vowel were then used by this subject for all subsequent studies which will be described later.

Figure 3 shows a narrow band sonagram of the vowel "Ha" on tone *G* (98 cps.). It is clearly seen how the vowel is composed of its characteristic formants in discrete frequency bands. The following Figure 4 represents wide band sonagrams. It illustrates the growing pitch of the fundamental tone by the increasing number of vertical striations per second which correspond to the glottal impulses; moreover, the tones sung in chest voice are much richer in higher harmonics than the higher tones a° and f^1, especially the latter in falsetto. Counting the vertical lines revealed that the tones were sung a trifle lower than prescribed by modern concert pitch which is tuned to a^1 of 440 cps. This was due to the fact that an old tuning fork of a^1 (435 cps.) was used as reference. These differences can be detected by a musical ear, but are negligible as far as laryngeal function is concerned.

5. Vocal Cord Tension and Vocal Pitch.

Everyone is aware that each human voice has two types of pitch. One is the *general pitch range*, or vocal compass,

to which the individual larynx is tuned by virtue of its dimensions and which determines the voice type. The other is the constantly fluctuating *pitch modulation* within the limits of the vocal range. These relative fluctuations of vocal frequency serve for the expression of emotional moods and of the vocal

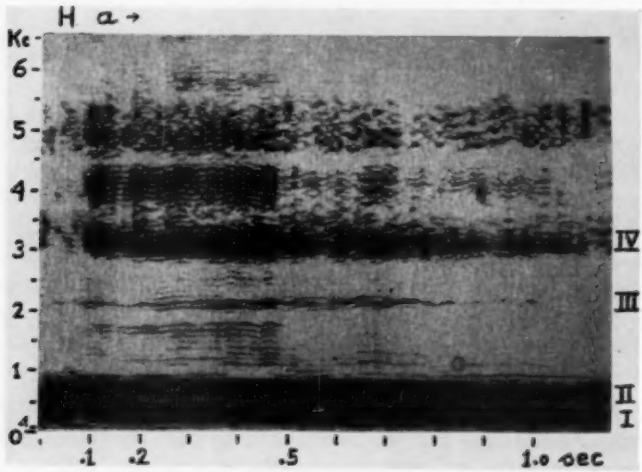

Fig. 3. **Narrow band sonagram** of vowel "Hah" sung on tone G (98 cps.) in chest register. The initial spirant noise of the "H" precedes the appearance of the characteristic four vowel formants marked I to IV.

elements of spoken language. For example, an interrogative sentence is characterized by elevation of the speaking pitch at the end of the phrase. Naturally, the absolute frequency values of both vocal range and pitch modulation are much lower in male or deep voices than they are in female or high voices; yet, the relative frequency ratios of such modulations may be quite similar among all speakers of a particular idiom and, therefore, serve to carry elements of information.

The *average speaking level* is easily recognized by matching it with suitable sound sources as mentioned before. In accordance with the constant fluctuations of the speaking voice, the so-called *prosodic melody* uses gliding intervals

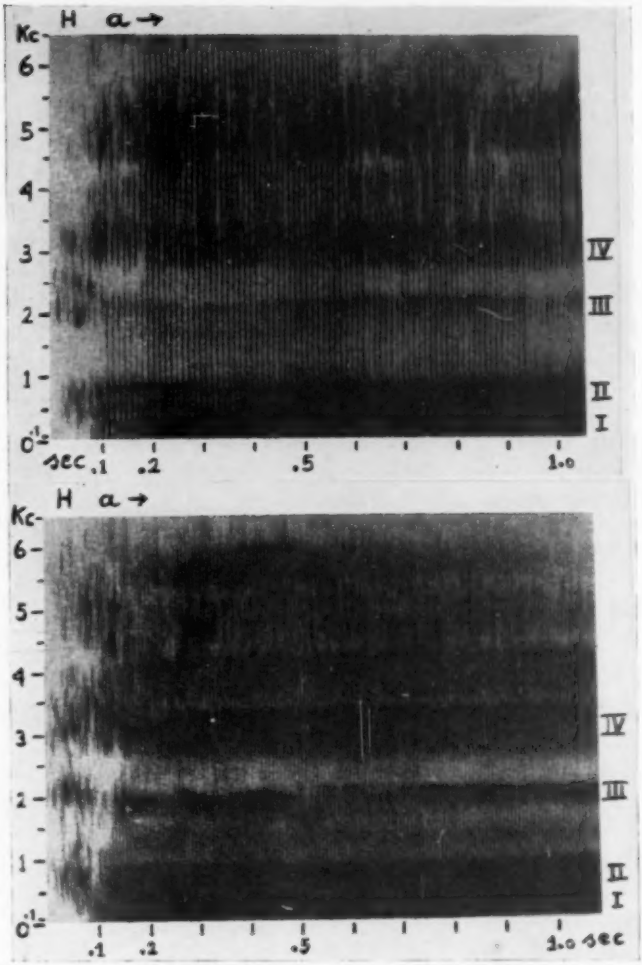

Fig. 4. **Wide band sonagram** of vowel "Hah" sung on four different pitch levels. a. Tone **G** (98 cps.) in chest voice, b. tone **d'** (147 cps.) at the lower register transition.

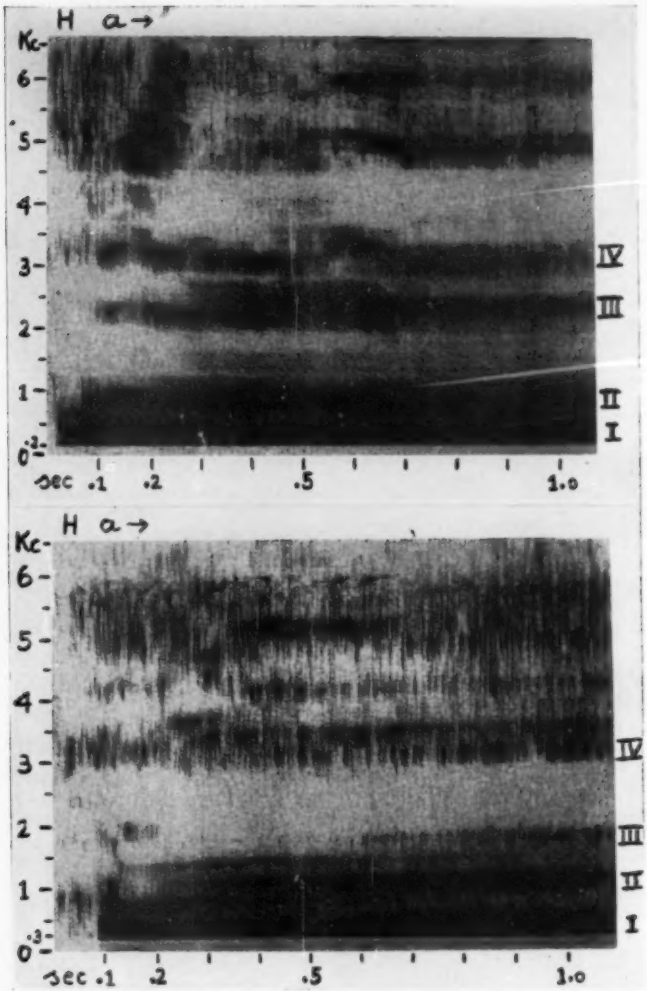

Fig. 4. c. Tone a^{o} (220 cps.) in middle register, d. tone f^{1} (350 cps.) in falsetto. The vertical striations correspond to the glottal impulses and their number per second indicates vocal frequency. Their count under magnification showed slightly lower frequencies than indicated due to lower tuning of the reference tone a^{1} (old fork at 435 cps.). Note spectrum of initial "H", increasing frequency of fundamental tone, and decreasing content of higher harmonics on the two highest pitch levels.

within half an octave. This speaking range is normally located near the lower border of the physiologic vocal range. This is why a low bass also speaks on low tones around 100 cps., whereas a little child or a petite woman will use a much higher speaking level around 300 cps. All voice experts agree that speaking on the most natural sounding or relaxed pitch level near to, but not directly at, the lower end of the vocal range, is optimal and least fatiguing. This physiologic speech level corresponds to the *neutral rest position* of the entire larynx.[75]

Cultural influences modify these physiologic rules to a large extent as can be heard daily in New York City. Puerto Rican girls, for instance, tend to speak with rather high pitch. Many American women, on the other hand, now find it desirable to speak at an artificially low level and which is often further lowered by chronic laryngitis resulting from smoking or vocal abuse (Compare subject II). Some radio announcers strive to use excessively low pitch levels in an effort to achieve a "booming" voice which is supposed to express reassuring masculinity and mercantile aggressiveness.

Since, then, each voice is determined in its *range* by the fixed physical dimensions of body and larynx, the *pitch modulations* of each voice within this physiologic range from the lowest to the highest available tone must be produced by a variable intrinsic mechanism of each larynx. This is the mechanism of vocal cord tension by the cricothyroid muscle. Since the classical experiments of Johannes Mueller (1839) it is well understood that the acoustic qualities of the voice are determined chiefly by the physical dimensions of the vibrating vocal cords; thus, we must distinguish between the absolute pitch range of each vocal organ and the relative pitch modulations it can produce within this range. The former depends mostly on the constant *absolute length* of the cords: basses generally have the longest cords, while the highest sopranos have the shortest. The latter is a function of variable elongation of the cords which modifies their *relative length* for each pitch level: within certain limits, vocal cord elongation increases with rising pitch.

Both principles follow general acoustic laws: one, the longer and heavier a vibrating system, the lower is its basic frequency range; two, the vibrating frequency increases with growing tension and elasticity. A functional compromise between these seemingly opposed principles is established by the mode of vocal cord vibration: with increasing pitch the *length of the vibrating glottis* becomes shorter and is shifted toward the anterior commissure; consequently, each pitch level is characterized by three values of length: *a.* absolute and constant cordal length, *b.* relative and variable cordal elongation, *c.* functional length of the vibrating glottis.

While these general notions are widely accepted, several details of the tensing mechanism require further clarification. In the first place, the question remains why there should be a register transition or break between the male middle and falsetto voice. It seems that the larger dimensions of the male larynx are naturally suited for the production of the low tones in chest and middle register; hence, high pitched phonation in falsetto register requires a *sudden shift in the vibratory mechanism.*[66] Equalization of these different modes of vibration and resonance by learned muscular adjustments in the entire coupled system of the larynx and its resonator (German: Ansatzrohr) is the goal of professional singing instruction in the various styles of Western vocal art.

Measurements of vocal cord elongation on high speed motion pictures from six male untrained subjects by Hollien and Moore[23a] revealed a different behavior of the cords in chest ("natural") and falsetto register. With increasing pitch, the cords lengthened in chest register until the register break was reached. For the higher falsetto tones, the cords seemed to shorten again in a slight but progressive degree. From these measurements the following conclusions are drawn:

a. Length of vocal cords increases systematically with rising pitch for the natural (chest) register; *b.* Vocal cords in abduction are longer than with phonation; *c.* No single pattern of elongation or shortening is apparent for pitches in the falsetto register; *d.* Magnitude of lengthening is no

greater in any one portion of the pitch range; *e.* There is some evidence of a "stair step" lengthening function.

6. Determinants of Vocal Pitch.

Numerous studies have produced a wealth of information on the dynamics of vocal cord tension. After the acoustic and stroboscopic characteristics of vocal registers were elaborated,[19,63,68,74] recent authors clarified various physiologic aspects by radioscopic and electrophysiologic studies.[8,25,48,75] It had been believed for a long time[35,36] that the falsetto register is produced by maximal contraction of the cricothyroid. Our myographic studies seem to confirm this opinion.

TABLE II.

Differences of Laryngeal Function in Chest and Falsetto Register of the Male.

	Chest Register	Falsetto Register
Shape of cords	rounded, full	sharp-edged, thin
Tension of cords	relaxed	tensed
Ventricular folds	adducted	abducted
Ventricles	narrow	wide
Glottal closure	complete in each cycle	spindle-shaped slit
Vibrations	broad, entire surface	narrow, marginal
Epiglottis	lowered	elevated
Supraglottic space	narrow	widened
Vocal pitch	low	high
Vocal timbre	full, resonant	thin, weak
Chief muscle	thyroarytenoid	cricothyroid

It is absolutely established that the cords *assume a characteristic shape* and a typical mode of vibration for each register.[8,9,29,31,49,62] Observed with stroboscopic illumination or with high speed motion picture techniques, the cords appear rounded, full, and relaxed for chest tones, while they are sharp-edged, thin, and taut for falsetto tones (see Fig. 10). At the same time, the cords vibrate broadly over their entire upper surface and close the glottis completely in the chest register, whereas in falsetto the vibrations are limited to the inner cord margins, and the glottis remains constantly open as a spindle-shaped slit. These marginal vibrations for the highest coloratura tones were seen as far as f^3 (1397 cps.) and g^3 (1568 cps.).[19] In addition, the ventricular folds partly

obscure the cords with chest tones, while they are more abducted in falsetto. Table II summarizes these characteristic differences of registers.

Although the sudden register break at the transition of middle and head register of the untrained voice suggests an abrupt change in laryngeal coordination at this point, the characteristic mechanisms of each register represent a gradual *series of intralaryngeal adjustments* (see Fig. 9). At the one end of this functional series may be seen the full and broad vibrations for the low tones in loud (forte), full, and resonant chest voice. On the other side, the series ends with the fine and shallow vibrations for the highest tones with weak (piano), thin, and faint untrained falsetto register. Between these two extremes are found the gradual steps of each tone across the middle register, the full sound of trained head voice, and the artistic falsetto.[48]

Many authors agree that loud intonation of a given tone shifts its register mechanism to the type of the next lower register, whereas soft intonation tends to raise this mechanism to the next higher type (see Figs. 9 and 14). This *mixture of function* is achieved by the ideal "voix mixte" technique of a well-trained voice. In a schematic outline, these gradual and overlapping transitions of phonic function may be aligned as follows: *a. Male voice.* Low chest tones: loud-soft; transition; middle register: loud-soft; transition; loud head voice (trained)—soft artistic falsetto (trained)—thin natural falsetto (untrained). *b.* In the *female voice,* the lower two registers behave similarly, while head voice can be only loud or soft and may be followed by a fourth flageolet register of highest coloratura voices.

All recent studies confirmed the influence of the cricothyroid muscle on vocal cord elongation and thus on the increase of pitch. Isshiki[25] enumerates three factors which determine the increase of pitch: *a. Vocal cord tension* by the cricothyroid; *b. Shape of the cord* as a result of thyroarytenoid contraction; *c. Vibratory mechanism* as determined by the configuration of the glottis. These three factors are

inseparately integrated with reciprocal interaction on the total vocal event.

According to Sonninen,[66] the cords are elongated for high tones by 4-5 mm. which means up to 25 per cent of their length for low tones. This elongation results in higher tension of the elastic vocal ligaments and increase of the distance between the vocal cord insertions; consequently, the external or *passive vocal cord tension* by the cricothyroid determines the cordal dimension as commensurate with each pitch level. Within this set frame of isotonic and crude cord tension, the isometric contraction of the vocalis muscle provides the finely graduated internal or *active tension* for the shaping of the cords and glottis as required by each type of intonation.

These complicated *interactions of several muscle systems* for each specific type of phonic function were duly considered in recent studies.[75] For the production of high tones, the cricothyroid must counteract the following factors: *a.* Increased muscular *activity of the vocalis* which grows three to sevenfold; *b.* Increased elastic *tension of the lengthened cords; c.* Increased *activity of the cricopharyngeus* which shortens the cords; and *d.* Increased *tracheal pull* resulting from laryngeal elevation. As the same authors[75] have shown, muscular activity of the cricothyroid depends on the spatial relationship of the cricoid and thyroid cartilages. Their EMG findings demonstrated increased cricothyroid activity when vocal cord tension was counteracted by other factors.

To test such additional factors, these authors[75] studied four stages of cricothyroid contraction. The subject was humming a high tone under the following conditions: *a.* Normal head position; *b.* Sudden opening of the mouth; *c.* Strong retroflexion of the head; *d.* Frontal pressure on the thyroid cartilage (Gutzmann's pressure test). As could be promptly heard, each procedure lowered the initial pitch of the tone stepwise. Simultaneous EMG recordings showed a corresponding *compensatory increase of cricothyroid activity.* These experimental findings explain the physiologic basis of Gutzmann's classical manipulation method for the suppression of habitual falsetto voice. Frontal pressure on the thyroid coun-

teracts the excessive contraction of the laryngeal elevators and of the cricothyroid which maintains the voice in the falsetto register.

LATERAL RADIOGRAPHY.

1. Cricothyroid Distance.

A few years after the discovery of X-rays, some physiologically inclined otolaryngologists availed themselves of this new technique for the study of laryngeal function.[31] In 1904, Moeller and Fischer[34] demonstrated the progressive narrowing of the cricothyroid space with rising pitch. This radiologic sign of cricothyroid function was later confirmed by many authors.[2,11,15,42,43,66] It is now understood that for the production of high tones the *cricothyroid distance is reduced to about 50 per cent* of its length with quiet respiration.

Figure 5 presents a lateral radiograph of vowel "ah" sung on four different pitch levels by subject I. As can be clearly seen the *cricothyroid distance diminishes* with rising pitch. It measured 17 mm. at rest, 16 mm. at tone *G* (98 cps.), 14 mm. at tone d° (147 cps.), 12 mm. at tone a° (220 cps.), and 10 mm. for the highest falsetto tone f^{1} (350 cps.). These values are very similar to those obtained in another male case studied some time ago.[2]

While the *relative ratio* of these measurements is typical, their *absolute values* depend on laryngeal size and, therefore, differ with sex, age, body type, etc.; however, the main finding in a normal case is the reduction of this distance with high tones to about 50 or 60 per cent of its value at rest. While this functional sign of cricothyroid activity is easily seen with fluoroscopy, it may not show as clearly on films when the cartilages are not yet calcified in young persons.

2. Vocal Cord Length.

Conversely, young individuals are best suited for the radioscopic study of *vocal cord length.* Laryngeal calcification in older persons obscures the exact dimensions of the vocal cords on various pitch levels as we have just seen on Figure 5. All one can determine on this illustration is a progressive length-

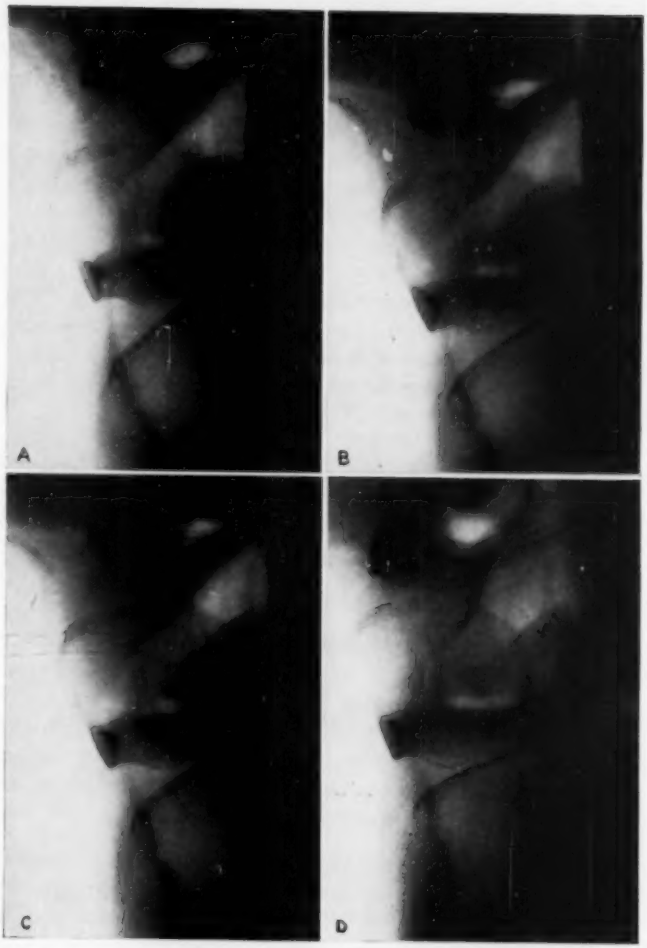

Fig. 5. **Radioscopic view of cricothyroid action** with rising pitch on vowel "ah". a. Chest register, tone G (98 cps.); b. transitional tone d' (147 cps.); c. middle register, tone a' (220 cps.); d. falsetto register, tone f' (350 cps.). Note increasing narrowing of crico-thyroid distance by elevation of cricoid arc.

ening of the laryngeal ventricle from 10 mm. at tone *G* (98 cps.), to 11 mm. at tone d° (147 cps.), 13 mm. at tone a° (220 cps.), and 15 mm. at tone f^{1} (350 cps.). It also appears that the cords become longer with increasing pitch, but this cannot be measured on this film.

Sonninen[66] has shown that a progressive lengthening of the vocal cords with rising pitch can be demonstrated on lateral radiographs of suitable persons. This increase in length may reach 4 to 5 mm. or 25 per cent of the vocal cord length for low tones; however, elongation of the cords is not strictly proportional to the ascent of a singing scale. After the maximum stretch has been reached at a certain frequency, vocal cord length remains the same, and further increase of vocal pitch is achieved by other principles, such as by the *mechanism of fixation isometry.*[66,69]

It appears that this change from the cricothyroid stretching mechanism to another principle of pitch increase is related to the register transition between the middle and falsetto registers of the untrained male voice. Very similar conclusions were drawn from the recent kinephotographic measurements by Hollien and Moore.[23a]

Various details of laryngeal behavior while producing different tones with specified techniques can be seen in a series of lateral radiograms taken in 1953 for another purpose. Hence, this second female subject cannot be compared directly with the first male test person on whom all other investigations were performed; however, the findings supplement the demonstration of various aspects of phonation.

Subject II: Normal healthy female, age 25, lyric operatic soprano with a delicately built and rather small larynx. *Vocal range:* d° (147 cps.) to f^{1} (350 cps.) in chest register, f^{1} to f^{2} (698 cps.) in middle register, f^{2} to d^{3} (1175 cps.) or e^{3} (1319 cps.) in head register. Both transitions at f^{1} and f^{2} are well equalized. This physiologic range is slightly larger than her musical range which extends from a° (220 cps.) to d^{3} (1175 cps.). *Average speaking range:* f° (175 cps.) to a° (220 cps.) in English, or a° (220 cps.) to d^{1} (294 cps.) in other languages.

This difference illustrates the influence of cultural factors on individual speaking habits.

Procedure: Irving Schwartz, M.D., took the lateral laryngeal radiographs at the New York Eye and Ear Infirmary for which gratitude is expressed. The subject phonated sustained tones on pitch level d^1 (294 cps.) in chest voice, a^1 (440 cps.)

TABLE III.

Radiographic Measurements of Laryngeal Dimensions as a Function of Vocal Pitch.

Tone	Rest	d^1	d^1	a^1	a^1	f^2	f^2	f^2	f^2	a^2	a^2
Cps	—	294	294	440	440	698	698	698	698	880	880
Volume	—	f	p	f	p	f	p	f	p	f	p
Method	—	open	open	open	open	open	open	*cover*	*cover*	open	open
Vowel	—	ah	ah	ah	ah	ah	ah	ee	ee	ah	ah
Hyoid horns level[1]	3a	2-3	2-3	2a	2a	2-3	2-3	2-3	2-3	2a	2a
elevation[2]	7	22	22	15	15	30	30	37	37	45	45
Epiglottis tip level[1]	3b	3c	3c	2-3	2-3	3c	3c	2b	2b	2b	2b
inclination[2]	67	52	52	45	45	52	52	67	60	60	52
distance to pharynx[3]	12	3	2.5	3.5	2.5	5	4	10	7	4	3
Glottis level[1]	5b	4-5	4a	4b	4b	4-5	4a	4b	4b	4b	4c
Ventricle height[3]	4	3	3.5	3	3	3	3	2	2	2	2
length[3]	8	9	11	13	13.5	16.5	17	17	17.5	17.5	16.5
Cord, length[3]	?	14	15.5	16	16.5	17.5	18	18	18.5	19	18

1. Re vertebrae.
2. In degrees.
3. In mm.

in middle register, f^2 (698 cps.) as the bridge tone, and a^2 (880 cps.) in head voice, always using the vowel "ah". In addition, the bridge tone of f^2 was also sung on vowel "E" (as in "me") to compare the equalizing technique of predominantly "covered" singing with "open" (or uncovered) singing; furthermore, each tone was first sung with high volume (forte, f) and then with low intensity (piano, p). Ten exposures of these four representative tones were compared with the situation at rest (quiet respiration).

Fig. 6. **Lateral radiograph** during quiet respiration. a. Outline of laryngeal structures.

Evaluation: As was learned from Sonninen's study,[66] the radiographic measurement of vocal cord length is at best a difficult and approximative procedure, because the fuzzy outlines of X-ray shadows are not easily delineated. To reduce the influence of measuring errors, the distances were measured with a caliper five times without comparing preceding results. No deviations greater than ± 1 mm. occurred. Averages were then entered in Table III.

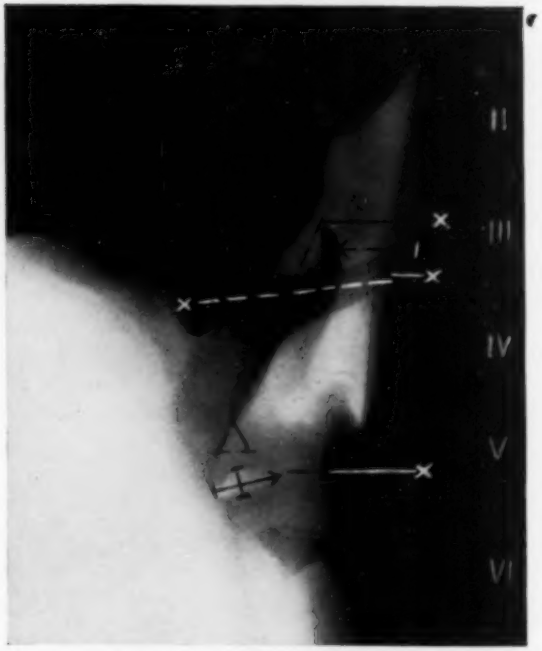

Fig. 6. b. Definition of measured dimensions and positions. Note resting position of all structures which differs significantly from their adjustments for various phonic actions. The posterior portions of the abducted cords are not visible.

Distances were measured as shown in Figure 6 which illustrates the larynx during quiet respiration. At first, the vertical changes in *laryngeal position* were related to the cervical vertebrae. Each vertebral body was defined by its number and subdivided into three portions: *a.* for its lower margin, *b.* for its center, and *c.* for its upper margin. For example, IIIb meant the midportion of the third cervical vertebra. A position nearest to an intervertebral space was indicated by the respective vertebrae, such as II-III for the second interspace. *Dimensions* of several laryngeal structures in mm. were then determined for each phonic event. Variable *inclination* of the hyoid horns and the epiglottis was indicated in angular degrees.

The *following structures* were observed: *a.* Level of hyoid horns; *b.* Elevation of hyoid horns; *c.* Level of epiglottis tip; *d.* Inclination of epiglottis; *e.* Distance from epiglottis tip to posterior pharyngeal wall, indicating width of laryngeal vestibule; *f.* Level of vocal cord; *g.* Height of laryngeal ventricle; *h.* Length of ventricle as far as visible; and *i.* Length of cord as far as visible.

Since the posterior ends of the last two structures were obscured by the ossification centers in the thyroid horns on the films of highest tonal frequency, one can be less definite about vocal cord length on highest pitch in this subject. It is also impossible to measure vocal cord length at rest on lateral radiographs, because the abducted cords form a posterior angle with the midline and, therefore, appear shortened.

Findings are summarized in Table III. As shown in Figure 7, rising pitch is characterized by *increasing elongation* of the cords and of Morgagni's ventricles. Within the examined vocal range, the cords lengthened by 5 mm. or about 30 per cent of their length at the lowest recorded tone. This increase was most marked for the lower frequencies in chest and middle register; however, it still continued to the highest recorded tone a^2 (880 cps.) sung in loud head voice. A slight shortening was noted with the same tone sung softly. Since the lowest and highest available tones were not recorded to spare the subject unnecessary exertion, no statements on the further behavior of her cords can be made. It is most likely that her cords were still shorter for her lowest tones, and it is possible that their maximal elongation decreased again for her highest tones.

The other structures described in the table followed a variable pattern of adjustment in relation to the *elevation of the larynx* and its parts. There was a distinct descent of the larynx at the transition tone f^2 (698 cps.). In addition, the spine became increasingly erect as the head was flexed forward with rising pitch. Such changes in position of the head are considered important by singers for the equalization of registers. Details of these variable adjustments will become clear with analysis of the following two illustrations.

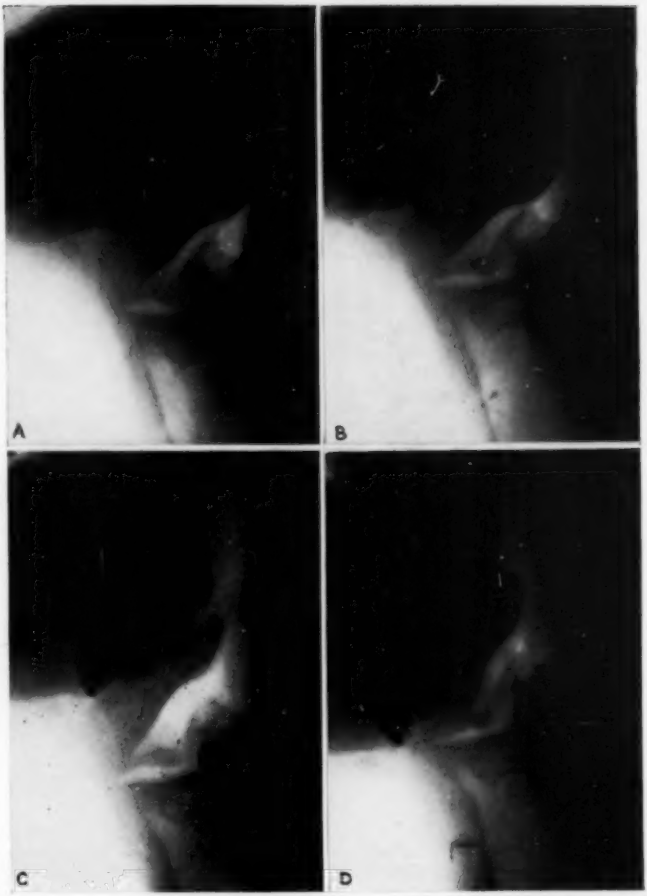

Fig. 7. **Lateral radiograph** of laryngeal adjustments as a function of increasing pitch, other vocal parameters being equal (vowel "ah", loud intonation, "open" singing). a. Tone d^1 (294 cps.), b. tone a^1 (440 cps.), c. tone f^2 (698 cps.), d. tone a^2 (880 cps.). Note increasing **elongation** of cords and ventricles, variable laryngeal **elevation** as expressed by changing height of epiglottis tip, hyoid horns, or glottis level, and related changes in **inclination** of epiglottis and hyoid horns. The spine becomes increasingly erect as the head is flexed forward.

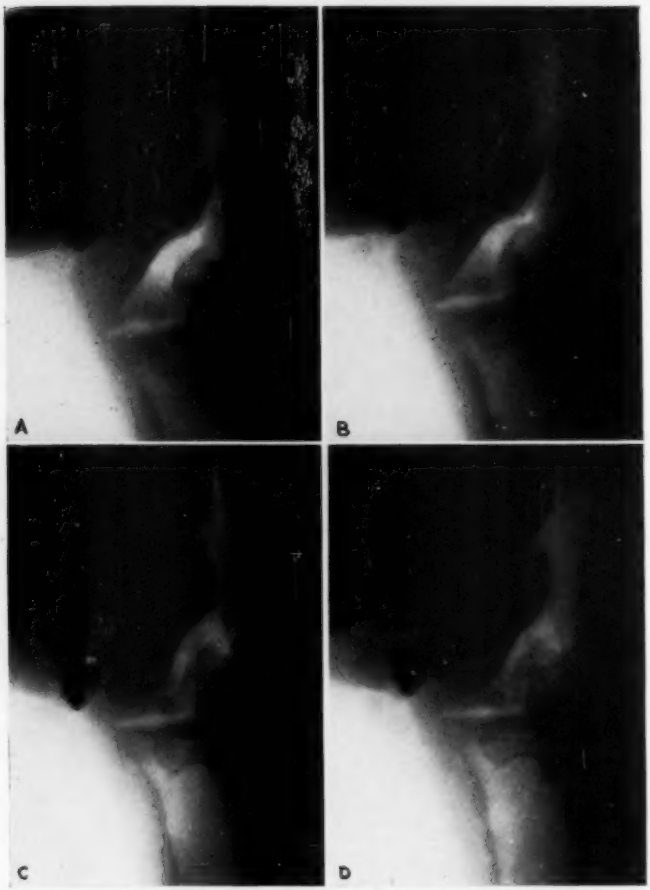

Fig. 8. **Lateral radiograph** comparing open and covered singing on equal pitch of transitional tone f^2 (698 cps.). a. Vowel "ah", open, forte; b. same, piano; c. vowel "E", covered, forte; d. same, piano. While length of cords or ventricles and level of hyoids remain about the same, the marked difference in vocal timbre is produced by the characteristic dimensions of the resonators: erection of epiglottis and widening of vestibule with covered tones.

In Figure 8 the additional vocal parameters of intensity and timbre are compared by open and covered singing of the same bridge tone f^2 (698 cps.). We now see further details of laryngeal adjustments to these different vocal actions. Vocal frequency remaining equal, there is little difference in the length of cords and ventricles. As will be discussed with Figure 9, a systematic difference was seen between *loud and soft intonation* in the sense that higher vocal intensity shifts the laryngeal mechanism to that of the preceding lower frequency level, and vice versa; however, an impressive change in dimension and configuration of the supraglottic resonator is clearly related to the contrasting techniques of singing an open vowel "ah" and the covered vowel "E". Without doubt, the *laryngeal vestibule is much wider with covered singing.* This supraglottic enlargement is due mainly to the elevation and erection of the epiglottis as has been known ever since laryngologists asked their patients to phonate a high-pitched and covered vowel "E" for best visualization of the cords.

All these interrelated laryngeal adjustments can be fully appreciated by examining Figure 9. Here, the various changes in laryngeal dimensions and positions are correlated with all three vocal parameters of frequency, intensity, and timbre. This illustration also gives an impression of the *gradual alterations of laryngeal function* for specific phonic performances. It can be seen again that the vocal organ must be studied as a whole system and cannot be dissected into isolated segments of function if serious errors of interpretation are to be avoided. This is like slicing an apple in various directions. Each slice shows another cross-section and it takes many such sections to present a composite reconstruction of the fruit.

Every well-trained singer knows that *certain phonic actions are more difficult* to perform well than others. Singing higher tones softly is harder than their loud intonation, the transitional bridge tones are not easily equalized, the highest tones require the greatest effort, and different vowels are given individual preference for specific combinations of the various vocal parameters. Let us see how the analysis of one subject's performance confirms these empirical facts.

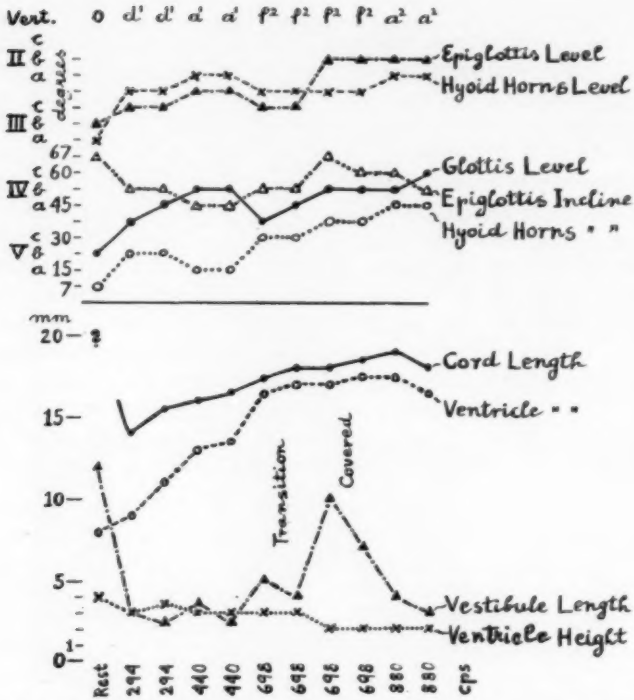

Fig. 9. **Graphic expression** of altered dimensions and positions of laryngeal structures in relation to the three vocal parameters of frequency, intensity, and timbre. Note contrast between regular increase in sagittal **elongation** with rising frequency and dromedary curves of vertical **elevation** of several parts which determine resonance. These spatial changes are most marked at the register transition and with covered intonation.

The following *conclusions* can be drawn from Figure 9 which are in perfect agreement with the experiences of vocal art and the findings of previous investigations[48,49]:

a. Laryngeal position tended to be elevated with rising frequency except at the upper register transition where it was lower as is the rule with most singers.

b. With *low vocal intensity* (piano) the glottis was higher, the cords and ventricles longer, and the laryngeal vestibule

more narrow than with loud (forte) intonation of the same tone and vowel. This shows again that soft intonation of a given tonal range approaches its laryngeal mechanism to that of the next higher tonal range in loud intonation and vice versa.

c. Covered production of a transitional tone at the upper register passage showed the epiglottis more elevated and erected, the hyoid horns more erected, the glottis at a higher level, cords and ventricles longer, and the laryngeal vestibule much wider than found with open singing of the same tone.

d. The *patterns of these laryngeal adjustments* in relation to the three vocal parameters are highly coordinated and intertwined in various directions, as can be seen from their manifold parallel or divergent progressions. From examining such graphic curves of laryngeal behavior one may gain some idea why good singing is so difficult an art which requires more endowment than just a healthy throat.

FRONTAL TOMOGRAPHY.

During the past 20 years, many authors employed this technique for the study of vocal cord configuration with various phonic functions. In particular, the problems of vocal registers, of covered versus open singing, and of vocal intensity could be clarified along the lines of accepted concepts. It can be clearly seen on all published tomographs that the well-known differences between the chest and falsetto registers (Table II) represent general characteristics of these vocal functions.[5,9,47,49]

As has been understood for a long time, these major differences demonstrate extremes of functional adjustments in the vocal mechanism. In between can be seen the graduated steps from one functional type to the next, such as the gradual transition along the series of chest, middle, and full head register, and their further extension to the soft artistic falsetto and the untrained thin falsetto.

Summarizing these findings, Berendes[9] concludes that *vocal intensity* on all pitch levels is correlated to the cross-sectional

shape of the cords. They are rounded and fuller in forte, and sharper and thinner on piano tones. *Register differences* between low and high tones correspond to the configuration of the vocal margin: While they are round and thick with low chest tones, they appear thin and sharp-edged for high falsetto tones. The vocal style of predominantly *open and covered singing* for the equalization of the upper register passage is also associated with similar functional adjustments of the cords: with open singing they are more rounded, while covering is accompanied by a thinner and sharper margin. All these accepted facts agree perfectly with the new findings explained in Figure 9.

Many authors believe that the full roundness of the cords for low, loud, and open tones results from increased *thyro-arytenoid* (vocalis) activity, whereas their thin and sharp-edged configuration for high, soft, and covered tones reflects increased *cricothyroid* activity.[9,49] As part of this study of cricothyroid function, we proceeded next to the tomographic analysis of the three tones previously explored on subject I with other methods.

Procedure: A. A. Adams, M.D., New York, took the tomographs in the following manner: 55 KV, 50 MA, supine position with the chin raised in order to remove the shadow of the lower jaw, 35 degree angle of rotation, 40 cm. focal skin distance, par speed intensifying screens, 75 milliampere/seconds. After a few trials, sections were taken on films at 8½ cm. A-P levels. Four valve unit, rotating anode tube with 1 mm. focus. The following four laryngeal functions were recorded: quiet respiration, and phonation of vowel "ah" on tone *G* (98 cps.), *a*° (220 cps.), and f^1 (350 cps.) with equal medium loudness.

Findings: Figure 10 demonstrates the typical tomographic appearance of a low, medium, and high tone in chest, middle, and falsetto register, respectively. One sees the increasing sharpening of the cordal margins, the enlargement of the laryngeal ventricles, the abduction of the ventricular folds, and the elongation of the supraglottic resonators, all in direct relation to increasing vocal pitch. All these changes in vocal

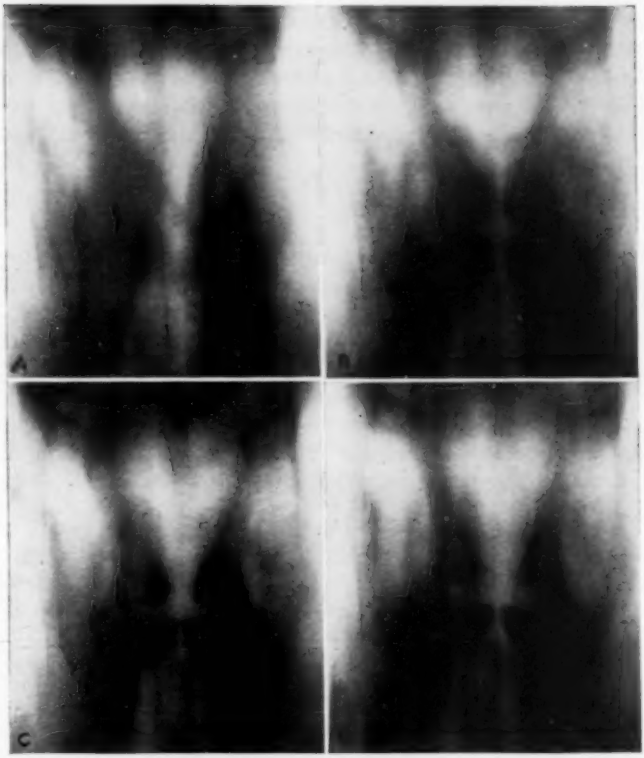

Fig. 10. **Tomographic view of vocal cord tension** with rising pitch on vowel "ah". a. Quiet respiration; b. chest register, tone **G** (98 cps.); c. middle register, tone **a**^{1} (220 cps.); d. falsetto register, tone **f**^{1} (350 cps.). Note increasing sharpening of vocal cord margins, widening of ventricles, abduction of ventricular folds, and elongation of supraglottic cavity.

cord configuration result from the synergic interaction between the *stretching cricothyroid* mechanism and the *inner tension by the vocalis muscle.*

ELECTROMYOGRAPHIC FINDINGS.

In recent years, the well established method of EMG has frequently been used for the demonstration of specific prob-

lems in laryngeal physiology and pathology. Theory and application of EMG were discussed by Lullies[63] and Faaborg-Andersen.[11,12] It seemed interesting to compare the EMG findings of cricothyroid function in subject I with the accepted opinions of previous authors and to elaborate certain details which should supplement the information on vocal cord tension.

1. Methodologic Limitations:

On the basis of previous investigations, it has become quite clear that laryngeal EMG is a reliable and refined method for the precise assessment of specific muscular actions as the basis of laryngeal functions. As long as the exact conditions and parameters of each experiment are clearly defined, the EMG records present objective data on the minute details of each functional element; however, this experimental *dissection of a global process* into elements of function also determines the methodologic limitations of such procedures.

The larynx is a most complicated motor organ whose manifold functions vary gradually between certain extremes of position and action. These variations are further influenced by numerous intrinsic and extraneous factors as we have already seen. It follows that all methods such as radiography, myography, etc., present an *instantaneous section* through one particular aspect of a fluctuating time sequence.

If a true representation of all interrelated factors were attempted, such experiments would have to utilize the *simultaneous recording* of the entire sequence of motor and acoustic events. This could be achieved by combining the following techniques: *a.* X-ray sound motion pictures of both lateral radioscopy and frontal tomography, *b.* high speed vocal cord film, *c.* electromyography, and *d.* sonagraphic recording of the vocal patterns. One of the purposes of this paper was to define what each of these methods can contribute to such analytic endeavors.

Work of previous authors[1,11-14,75] has shown that laryngeal EMG in a conscious human is not at all easy. Since the extrinsic and intrinsic muscles of the larynx are subject to

many individual variations of development, the placement of needles is complicated by such factors as neck configuration, amount of adipose tissue, body size, etc. Unless one resorts to surgical exposure, one can never be absolutely positive about reaching a certain muscle. Such technical difficulties may account for some differences of results as described in the literature.

2. Accepted Facts:

All observers agree on the presence of constant *resting potentials* during silent respiration. Buchthal[12] described these potentials as "proprioceptive postural activity." According to Fink, Basek, and Epanchin,[14] the continuous balanced activity of the intrinsic laryngeal muscles seems to be necessary to stabilize the cricoarytenoid joints. The pattern of activity in the extrinsic muscles indicates their major role in controlling the lateral excursion of the arytenoids and thus the width of the glottis. These resting potentials are seen in our Figures 11, 12, 13, and 15.

It has been known for a long time that during quiet respiration the vocal cords are in constant *pendulous motion around their neutral or resting position* which is marked by the intermediate line.[30,67,70-72] As can be seen during laryngoscopy, and even in the course of laryngectomy as long as the nerve supply remains intact, the cords are slightly abducted with inspiration, and slightly adducted with expiration. It is generally held that these pendulous movements reflect the undulating tonic activity of the intrinsic muscles.[71] Inspiration is thus accompanied by increased tonus of the abducting posterior cricoarytenoid and inhibition of the adductors. The reverse occurs in expiration.[38,39] Obviously, this alternating tonic activity is under the influence of the regulating mechanism of the bulbar respiratory center which, in turn, follows the respiratory reflexes mediated by the various chemo-receptors.

In view of these physiologic facts, it was not surprising to learn from Murtagh and Campbell[56a] that EMG reflected the same type of undulating muscular activity. They showed

that the abducting posterior cricoarytenoid was more active during inspiration, while the *adducting* cricothyroid and vocalis muscles increased their activity with each *expiratory* cycle. This finding agrees with the observations of older authors[22,38,39,67] who found active contraction of the cricothyroid with expiration, and its relaxation with inspiration. Krause[38] called the cricothyroid an expiratory muscle which initiates phonation by its contraction and elevation of the cricoid ring. Unfortunately, this question seems to be more complicated because other authors came to different con-

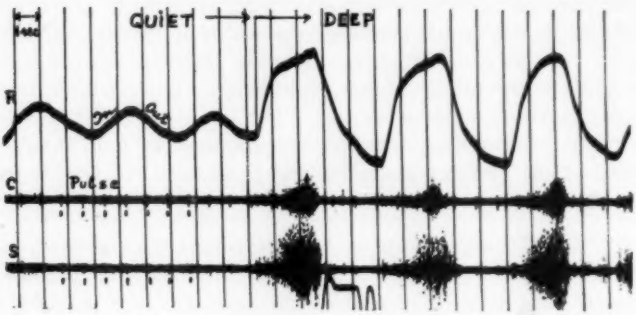

Fig. 11. **EMG record of silent respiration.** R: Pneumogram, C: Cricothyroid muscle, S: Sternothyroid muscle. Note cardiac pulse and compare the slight constant resting activity of these muscles during quiet respiration with their marked activity in deep respiration. Their maximal activity coincides with the height of inspirium and ceases sharply with beginning expiration. This time interval expresses the latent period of muscular activity.

clusions.[11] Since this paper deals only with the cricothyroid muscle, we need not discuss this problem any further. Suffice it to state that our records contribute the following data to this question.

Figure 11 shows that no activity in the cricothyroid and sternothyroid was recorded with *quiet* respiration. In *deep* respiration, both muscles are very active at the height of the inspirium, or about 0.5 sec. before expiration sets in. This period agrees with the latent time noted by other investigators.[11] The same latent period is seen on Figure 12 where

a maximal expiratory effort is accompanied by marked activity in both muscles. Figure 14 demonstrates the expiratory function of the cricothyroid very clearly. Its maximal activity occurs with a latent time of about 0.3 to 0.5 sec. before the onset of each intonation of high tones. The same expiratory activity is seen in Figures 15 and 16, if the latent time of 0.3 to 0.5 sec. is taken into consideration. These records, therefore, lend weight to the clinical assumption that the *cricothyroid is an expiratory muscle.*

All authors are in agreement on the characteristic existence of a *latent time* between the first visible appearance of the action potentials and the subsequent audible onset of a phonic function. Accordingly, electrical activity begins before actual phonation. For the cricothyroid muscle, the latency was measured as 0.1 sec. (Lullies[63]), while Faaborg-Andersen[11] found a time interval of 0.35 to 0.55 sec. in all investigated adductor muscles.[75] Similar values can be seen in our recordings as just explained. This phenomenon represents objective evidence for the old concept of "inner singing" because it also occurs with mere imagination of a tone to be sung without actually producing it; thus, the laryngeal muscles bring the cords into proper position and tension for each vocal performance before the expiratory air stream begins to set them into vibratory motion.

In view of the recent controversy about the validity of the classical myo-elastic or aero-dynamic theory of passive and tonic vocal cord vibration versus Husson's new cerebral or neuro-chronaxic theory of active and clonic vibration,[31,49] it is interesting to note the maximal *discharge frequency* of single motor units. Lullies[63] reports Katsuki's findings which showed that the cricothyroid muscle begins to be active above 150 cps. of the male voice in the form of single unit potentials of 2-3/sec. (Comp. Fig. 15). With rising pitch, the discharge frequency increases until 20-40 impulses per second are produced for the highest tones of falsetto voice. Faaborg-Andersen[11] described a similar discharge frequency of a single motor unit ranging from 3 to about 30 per sec. These discharge frequencies are, of course, much lower than the vibra-

tory frequencies in cps. of the audible tones (see Figs. 15 and 16).

There cannot be the slightest doubt about the function of the cricothyroid muscle as the primary and external *vocal cord tensor*. All previous findings by lateral radiography, frontal tomography, EMG, and direct surgical observation demonstrated this action very clearly. When a gliding scale was sung from 167 cps. to 358 cps. by a normal man,[11] the cricothyroid showed an increase in maximal amplitude of action potential pattern by 200 to 600 per cent. The same increase during glissando humming upwards over the entire vocal range was recorded by Zenker and Zenker.[75] They concluded that the tensing cricothyroid shows increasing activity with rising pitch, whereas its antagonist, the relaxing cricopharyngeus, is very active during any type of phonation, but predominantly with the low tones.

Since phonation within the average speech range is nearest to the neutral rest position of the entire larynx,[75] speaking pitch requires little cricothyroid activity. This reciprocal antagonism of the cricothyroid and cricopharyngeus seems to explain the long-sought external and isotonic *frame-tension* of the vocal cords within which the inner and isometric tension by the vocalis muscle permits the fine regulation of glottal shape for the differentiation of registers and timbre, as shown in the radiographic illustrations Figures 7 to 10.

While all these findings demonstrate the *general pattern* of cricothyroid activity, no attempts have yet been made to correlate its function with *specific frequencies* over a larger tonal range; therefore, we supplemented our investigations of cricothyroid function with the EMG study of the same subject I. In addition to the vocal events as defined above (see Figs. 3 and 4), we also analyzed several other phonic and respiratory phenomena. This experiment was conducted in the Department of Anesthesiology, College of Physicians and Surgeons, Columbia University, with the help of Raymond Fink, M.D., and Milos Basek, M.D.

3. Technique.

After many trials, insulated unipolar needle *electrodes* with

a bare point were inserted through the skin into the sternothyroid and cricothyroid muscle on the right side. The sternothyroid electrode was located just behind the *linea obliqua*. The cricothyroid electrode in the *pars recta* recorded the strongest potentials when penetrating to the inferior border of the thyroid cartilage near the midline. A common indifferent electrode was located subcutaneously in the left shoulder.

The *EMG potentials* were passed through a band-pass filter with a lower limit of 100 cps. and an upper limit of 2500 cps. The potentials were monitored on a multichannel oscilloscope and recorded cinephotographically.

A *pneumograph* around the lower chest was used to signal the phases of respiration. The changes in pressure in the pneumograph were detected with a Statham strain gauge and recorded on the upper channel of the oscilloscope. Polarity was arranged in such a manner that inspiration produced an upward deflection as is customary with phonetic investigations.

4. Experimental Procedure.

After insertion of the needles, subject I performed the following actions: *a. Quiet respiration:* in-out, three times. *b. Deep respiration:* in-out, three times. *c. Phonation of single tones* on vowel "ah": tone *G* (98 cps.), weak volume, three times, loud volume, three times; tone d° (147 cps.), weak volume, three times, loud volume, three times; tone a° (220 cps.), weak volume, three times, loud volume, three times; highest available tones f^{1} (350 cps.) to g^{1} (392 cps.), weak volume, three times, loud volume, three times. *d. Quiet respiration,* as before. *e. G major scale* over two octaves from *G* (98 cps.) to g^{1} (392 cps.) and back, each tone sung separately like "ha - ha - ha" for definition of exact length of each tonal step. A register break occurred at tone d^{1} (294 cps.). *f. Quiet respiration,* as before. *g. Respiratory efforts:* deepest silent exhalation, Valsalva, Mueller, prolonged silent expiration, sucking, swallowing, and whistling, each three times.

5. Findings.

As is well known from radiologic findings (see Fig. 5), the cricothyroid is at rest during quiet respiration. No fluctuations in its postural resting activity could be picked up with our procedure (see Fig. 11). The heart beat is clearly seen in the leads from both muscles. In *deep* respiration, marked activity occurs in both muscles, with a latent maximum about 0.3 to 0.5 sec. before onset of expiration. This is the latent time.

Figure 12 shows an interesting fact. With *maximal* expira-

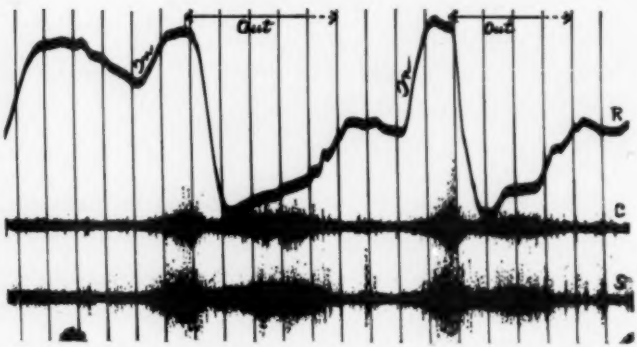

Fig. 12. **EMG record of maximal expiratory effort** (vital capacity). Abbreviations as before. Note increased muscular activity during deep inspiration and with the second phase of the expiratory effort.

tion (vital capacity), there is marked activity shortly before expiration. In the first phase of rapid exhalation, both the cricothyroid and the sternothyroid reduce their activity; however, during the second phase of forced expiration, both muscles participate in the expiratory effort which is demonstrated by the ascending pectoral curve. These findings again tend to confirm the expiratory activity of the cricothyroid.

Figure 13 offers proof for the correct placement of the needles. It shows three periods of *labial whistling*. Since this phonic action proceeds without laryngeal phonation, the vocal

cords are at rest and the *cricothyroid* remains inactive; however, the larynx moved slightly upward in this subject, which is expressed by the activity of the *sternothyroid.* Apparently, the depressing function of this muscle comes into play as soon as the laryngeal elevators require the antagonistic collaboration of the laryngeal depressors. It is noteworthy that activity in the sternothyroid begins about 1.0 sec. before the onset of labial whistling.

In the experiment of *singing separate tones* on various pitch levels (see Fig. 14), the increase of *cricothyroid* activity with

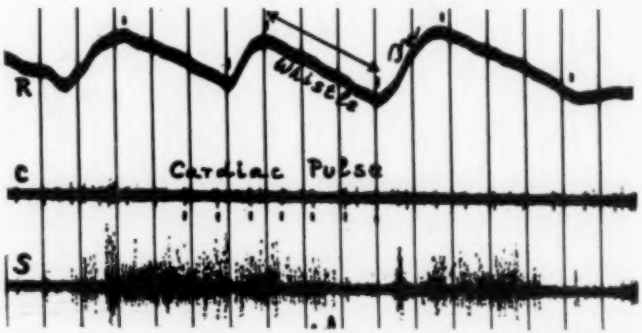

Fig. 13. **EMG record of labial whistling.** Note absence of **cricothyroid** activity because the vocal cords are at rest. Marked **sternothyroid** activity reflects the vertical laryngeal movements during this expiratory action.

rising pitch is well seen. Each pair of records represents first soft (piano) and then loud (forte) intonation. It is obvious that loud volume requires greater muscular activity than does weak volume. Again, a first burst of action potentials appears about 0.5 sec. before phonation. In perfect agreement with previous findings,[11,14,63] cricothyroid activity begins at the tone d° (147 cps.) of this male voice. A new, though typical finding in our records is a notch (x) in the respiratory curve immediately preceding phonation. This notch is visible in all four examples of loud intonation, especially of the higher tones. It represents the additional expiratory effort which is

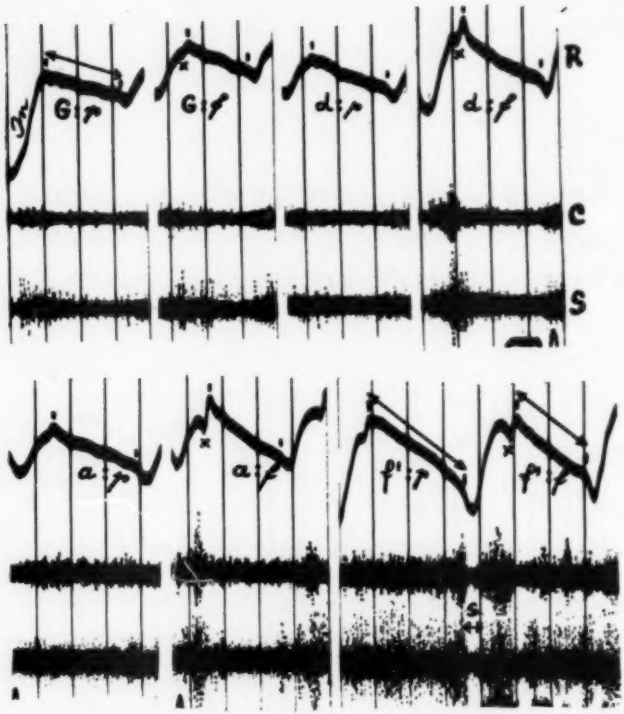

Fig. 14. **EMG record of singing single tones** of varying pitch on vowel "ah", male, baritone. p= softly (piano), f= loudly (forte). Tone **G** (98 cps.) piano and forte, tone **d°** (147 cps.) piano and forte, tone **a°** (220 cps.) piano and forte, tone **f¹** (350 cps.) piano and forte. **Cricothyroid** activity begins at about 150 cps. (middle register) and reaches its maximum with the highest available falsetto tone. **Sternothyroid** activity begins with the lowest chest tone and increases with rising pitch. Both muscles are more active with loud tones. Note typical notch (x) in respiratory curve preceding loud intonation as a sign of respiratory support (appoggio).

known to singers as active *breath support* or "appoggio" of loud and high tones.

In contrast, *sternothyroid* activity is visible throughout all these recordings of separate tones (see Fig. 14). It sets in about 0.7 sec. before intonation of weak low tones and increases with rising pitch and higher intensity. Note interval of electrical silence at point (S) immediately after cessation

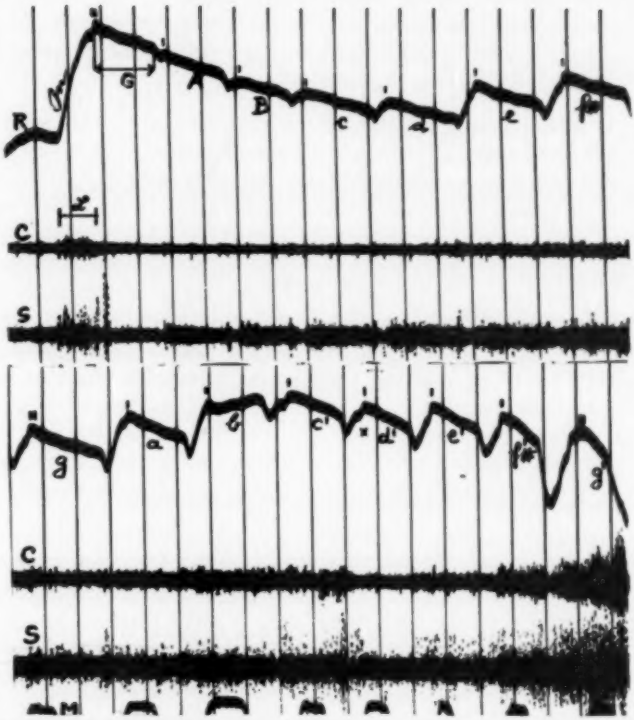

Fig. 15. **EMG record of G major scale** sung upwards medium loudly (mf) over two octaves from **G** (98 cps.) to g^1 (392 cps.). The small vertical lines indicate the onset of each tone, the double lines indicate octaves. A register break between middle and falsetto voice occurred at d^1 (294 cps.), marked (x). The **cricothyroid** is active in the inspiratory latent period (L) before phonation and begins to contract above tone d^0 (147 cps.), reaching a maximum at g^1 (392 cps.). The **sternothyroid** is more active throughout the entire record, reaching maxima during the inspiratory latent period, on tone e^1 (262 cps.) before the register break, and with the highest tones g^1 (392 cps.). M= marks for identification of tones.

of tone f^1 (350 cps.) with soft intonation, lasting 0.4 sec. until the next deep breath is taken for loud phonation of the same tone. Although both muscles often show simultaneous activity, the patterns of their action potentials are distinctly different. When considering the characteristic latent time in the appearance of potentials before the onset of phonation, it is again clear that both muscles are most active during expira-

tory phonation. *Both muscles* are more active with loud (forte) intonation which reflects the greater muscular effort. This fact should be studied in relation to the radiologic findings shown in Figures 8 and 9.

As a final demonstration of the gradual increase in activity of the *cricothyroid* with rising pitch, Figure 15 shows the record of the *ascending G major scale* over two octaves. With a latent time (L) of one second before intonation of the first tone, both muscles increase their activity as a sign of preparatory "inner singing" while the larynx and the cords assume their position of phonation; however, there is no increased cricothyroid activity throughout the chest register. It is quite obvious that cricothyroid contraction becomes visible with tone d° (147 cps.) at the passage to the middle register and then increases until tone d^{1} (294 cps.) where a *register break* (x) marked the abrupt jump into the falsetto register of this untrained baritone voice. The highest tones until g^{1} (392 cps.) show strongest activity.

A similar pattern is produced by the *sternothyroid* muscle which also shows the initial latent activity (L) with inspiration for the first tone; however, it is active throughout the entire scale, ending each tonal step with a distinct burst of activity before the next breath is taken. At the *register break*, the sternothyroid is very active as a sign of the sudden change in laryngeal position (Comp. Fig. 9). The highest tones are accompanied by great activity which indicates the general laryngeal effort.

Figure 16 is the direct continuation of the preceding illustration, showing the same G major scale *sung downward* over two octaves. All phenomena observed with the ascending scale are now seen in the reverse order. The vocal inequality at the *register break* (x) is expressed by increased activity in both muscles. One striking difference in the descending scale is the continuing activity in the *cricothyroid* with low chest tones. After its continuous contraction diminished down to tone d° (147 cps.), there remain discrete bursts during the latent time of each of the last four chest tones. This phenomenon of different cricothyroid activity with ascending and

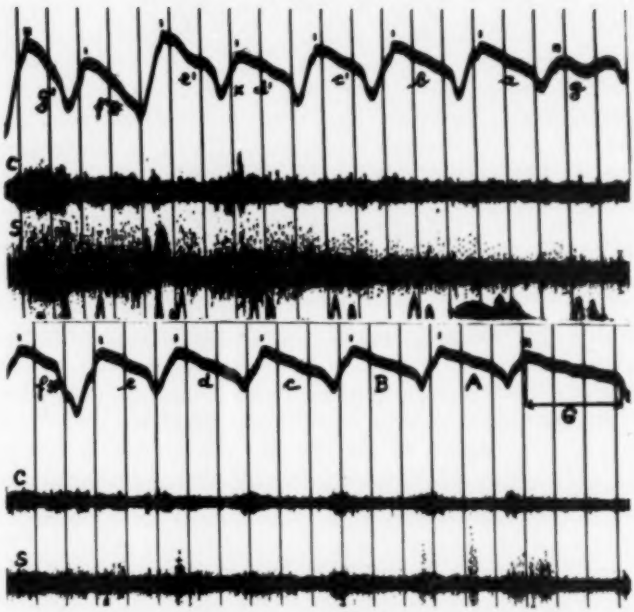

Fig. 16. **EMG record of G major scale** sung downwards medium loudly (mf) over two octaves from g^1 (392 cps.) to G (98 cps.). Tones and octaves indicated as before. The **cricothyroid** is most active with the highest tones and at the register break (d^1), and shows decreasing activity until tone d^0 (147 cps.); however, it remains active with decreasing pitch, contracting also during the inspiratory latent period of the lowest chest register tones. The **sternothyroid** is most active with the highest tones and at the register break (x), and less active with the lowest frequencies.

descending pitch changes deserves further investigation. A final burst of activity in the *sternothyroid* for the lowest tone *G* (98 cps.) represents the lowered laryngeal position before the vocal organ comes to rest at the end of this performance.

6. Comment.

These patterns of action potentials are *generally characteristic* for the investigated vocal functions. In particular, the cricothyroid shows its increasing activity with rising pitch. This was to be demonstrated.

The question remains at what frequency the cricothyroid begins to show increased activity. Our findings agree with previous reports,[11,63,75] indicating that this muscle begins to be active above 150 cps. of the male voice. Since the majority of males have a baritone voice, these observations seem to be valid for this particular voice type. If we recall that the tones d° (147 cps.) or e° (165 cps.) mark the transition between chest and middle register of the baritone voice, it would follow that cricothyroid activity sets in with the *transition to the middle register* and increases to a maximum with the falsetto register.

Providing these assumptions can be corroborated by further studies of all voice types, cricothyroid function would help to explain the *intrinsic* portion of the register mechanism. Its *extrinsic* portion is obviously correlated with laryngeal positioning by means of the external strap muscles. Radiologic indications of this extrinsic portion were shown in Figures 8 and 9.

Other details, such as the *register break*, illustrate the *individual performance* of this untrained male voice on the selected sequence of vocal events. Different details are to be expected with different voices, especially with trained singers, and with different vocal performances.

It is clear that EMG picks up many fine details of laryngeal function which reflect the audible differences in vocal performance. Further studies of precisely defined vocal phenomena with such methods will reveal additional details of the highly complex process of phonation. If each vocal action is suitably isolated from the complicated time sequence of singing, many characteristic elements can be defined as part of the total and integrated event.

The problem of experimental isolation of specified functions is one reason why the fine details of EMG findings reflect certain differences in the observations of various authors.[12a] As more investigations of this type will become available, the general laws of laryngeal physiology will be more clearly understood. It will then be possible to differ-

entiate the constant *general trends* of laryngeal movements from the variable *individual habits* of single persons.

LARYNGOSCOPIC FINDINGS IN CRICOTHYROID PARALYSIS.

1. Clinical Occurrence.

Laryngologic textbooks traditionally illustrate the condition of cricothyroid or superior laryngeal nerve paralysis by a wavy or serpentine margin of the affected cord. This theoretical construction has never been visualized in clinical practice for the simple reason that it does not exist,[2,3,5,9,20] yet, this old misconception confuses the practitioner who will not think of cricothyroid paralysis as long as he believes he sees straight cords. For this reason, the condition of isolated paralysis of the superior laryngeal nerve or of its end organ, the cricothyroid muscle, is very rarely recognized.

Several authors remarked on this discrepancy between the frequency of clinical occurrence and recognition in practice.[43,50] When discussing some surgical considerations of the superior laryngeal nerve, Gregg[21] quoted Bowles who stated that paralysis of this nerve after thyroidectomy "is far from a rare occurrence." The external motor branch especially is easily injured during this operation.[55] Gregg[20] also confirmed the *non-existence of serrated cords*[5] when he severed both superior laryngeal nerves in dogs; there was no change in vocal cord motility nor in the contour of their margins.

Mygind,[57] in 1906, was the first to describe the clinical syndrome of *cricothyroid paralysis.* His main findings were: *a.* laryngeal hemianesthesia resulting from involvement of the internal branch of the superior laryngeal nerve; *b.* oblique glottis as a sign of cricoid torsion from unilateral muscle contraction; *c.* absence of narrowed cricothyroid distance on lateral radiographs while attempting high tone phonation.

Etiology: Since then further observations have added to the understanding of this condition which is now recognized as a specific syndrome that may be caused by various pathologic events. With regard to etiology, the most frequent cause is *thyroidectomy.*[2,10,32,37,42-45] In previous years *diphtheria* was

a common peripheral cause.[43,49] *Injury*, from accidents or in times of war, may damage either the nerve or the muscle directly.[2,49] Sometimes, a mild and transitory damage to the muscle may result from extreme *vocal overexertion* (screaming in panic), or from violent vomiting. I once saw a famous opera soprano who could not sing high tones for a few days and had pain in both cricothyroid muscles following a gastric disorder with violent vomiting. Cases of *central origin* range from vascular accidents,[50] degenerative brain disease[46] or brain tumor[40] to syphilis.[46]

2. Laryngeal Anesthesia.

When suspecting a paralytic lesion of the larynx, the first sign to be considered is anesthesia of the upper laryngeal space. Touching the various parts with a fine probe will promptly reveal whether or not sensibility is reduced. If anesthesia is found, involvement of the *inner* branch of the superior laryngeal nerve is established. This may be unilateral or bilateral, partial or complete, and will then help to localize the level of the lesion; moreover, the patient may be subjectively disturbed by laryngeal *paresthesia* or *neuralgia* in the region of the superior laryngeal nerve. On the basis of such sensory disorders, *choking spells* may interfere with meals and sleep.

Anesthesia is most likely present in cases of *central* origin[40,44,46,50,51]; however, it may also result from *low vagus lesions* in the chest due to retrograde degeneration of the entire nerve trunk up to the motor nucleus ambiguus in the medulla with secondary loss of all motor and sensory innervation by the vagal branches.[23] In *peripheral* cases, anesthesia may or may not be present, dependent on whether the lesion, such as by thyroidectomy, affected the inner branch.[2,10,42,43]

If, then, laryngeal anesthesia is found, paralysis of the superior laryngeal nerve is quite likely. Its absence, however, does not rule out such paralysis, for the external motor branch alone, or the cricothyroid muscle itself may be the seat of the lesion which then leaves the internal sensory branch intact.

3. Laryngoscopic Appearance.

Paralysis of the superior laryngeal nerve or of the cricothyroid muscle is easily overlooked, because the visible changes in the affected cord are not too conspicuous; however, the experienced and alert observer will recognize the typical alterations of glottic appearance when he thinks of this possibility.

Oblique glottis. Many authors[2,10,37,42,57,62,67] agree on the characteristic sign of a deviated glottal axis in unilateral paralysis. As is well known, the glottis may be oblique due to several causes, namely, *a.* congenital asymmetry of the entire larynx,[49] *b.* torsion of the entire larynx from pressure by an enlarged thyroid gland, or *c.* torsion of the cricoid cartilage in relation to the thyroid.[2] The latter is caused by unilateral inactivity of one cricothyroid muscle.

As shown by numerous stimulation experiments, and as explained in Figure 2, unilateral contraction of one cricothyroid muscle rotates the cricoid ring anteriorly to its side so that the *posterior commissure deviates to the side of paralysis.* It is important to describe this posterior deviation in order to avoid confusion of sides and directions. Otherwise, conflicting interpretations will continue to complicate the description of such findings. Absence of glottic deviation by no means rules out cricothyroid paralysis, because it cannot occur in cases of bilateral involvement; hence, the oblique glottis is an important sign of unilateral paralysis.

Lack of tension in the affected cord. Since the cricothyroid is the main external tensor of the cords,[29,61] its *unilateral* paralysis leads to loss of external tension in the affected cord.[2,10,37,40,49,50,60,69] As a result, the paretic *cord will be shortened by the intact vocalis muscle.* Under this pathologic condition, thyroarytenoid action can no longer be isometric and, therefore, shortens the cord by abnormal isotonic contraction.[75] This reduction in length may be more pronounced during high tone phonation when the other cord is normally lengthened by unilateral cricothyroid contraction. It means the same when several authors described the afflicted cord

as flaccid, loose, or flopping. No such differences in length may be expected in cases of *bilateral* paralysis, because both cords will then be equally relaxed and flaccid.

Level differences of the affected cord. In agreement with the accepted loss of tension, many authors described the paretic cord as hanging down on a lower level.[29,44,51,60] Our Figure 1 shows why *unilateral* action of the intact cricothyroid should elevate the same side of the cricoid[67] and thus *lower the affected cord* with phonation; however, there is a second factor involved. Resulting from the flaccid loss of tension, the affected cord is aspirated to a sagging *lower level with inspiration* and blown up to a bulging *higher level with expiration.*[50,62] The interaction of these two factors determines the actual appearance during a given vocal performance. This explains variable observations of different authors. Again, no such level differences should be expected in *bilateral* paralysis.

Hyperemia of the affected cord. According to electrophysiologic experiments,[16,20,62] stimulation of one superior laryngeal nerve produces tensing and blanching of the corresponding cord. It is logical to find hyperemia of the affected cord with paralysis of the entire superior laryngeal nerve as Kecht[37] actually observed in a postoperative case. Possibly, interruption of the sensory internal branch may be responsible for this finding on the ground that sympathetic vasoconstrictor innervation is then lost; however, this sign is not constant, and its relation to altered blood supply following thyroidectomy is not clear.

Prolapse of respective arytenoid. As Stupka[67] has shown with electrical stimulation, unilateral cricothyroid action causes the same arytenoid to tilt backwards. Conversely, cricothyroid paralysis should be followed by arytenoid prolapse on the same side. This was indeed seen by several authors[46,50] and by me since paying attention to this fact. There are other descriptions which noted the opposite behavior.[2,49] This sign will, therefore, require further study.

Additional findings may be produced by the pathologic

process in the individual case. Lawson[40] described a case of central origin which was verified as a tumor of the left temporal lobe. While vocal cord adduction was not reduced, the presence of laryngeal anesthesia provoked *choking spells and adductor spasm.* The voice was typically feeble, low, rough, and easily fatigued. As a rule, such disorders of laryngeal reflex function originate from involvement of the sensory internal branch of the superior laryngeal nerve.

PHONIATRIC ANALYSIS OF VOCAL SYMPTOMS.

The main vocal symptom of cricothyroid paralysis in man was well known to the older authors: the voice is low, weak, and rough. In experimental confirmation, section of one or both superior laryngeal nerves, or excision of the cricothyroid muscle in dogs was often found to produce a comparable hoarseness of the bark tone.[33,58]

After Mygind[57] had given the first complete description of the *laryngeal* findings in cricothyroid paralysis, Luchsinger[42-46] elaborated the *vocal* symptomatology of this condition. His findings were soon confirmed and augmented by others.[2,9,10,37] By comparing these descriptions with personal observations, we arrive at the following definition of the audible signs. Although clearly present in cases of unilateral paralysis, all these signs are more marked in bilateral afflictions.

1. *Vocal weakness* is the main subjective complaint. Objectively, the voice sounds feeble, lacks in resonance and projection, and becomes quickly fatigued after brief efforts. Raising the voice, such as for calling aloud, is obviously impossible to the patient. These findings are readily explained by the loss of tension in one or both cords. Owing to its flaccidity, the relaxed cord is blown upward or outward by the expiratory air stream. Consequently, glottal closure becomes ineffective and part of the phonatory air is wasted.

2. *Shortened phonation time* presents a direct measurement of the phonatory air waste. All observers stressed the marked reduction of phonation time from a normal value between

20 and 30 sec.[2,4,31,49] to highly pathologic values of only 5 to 10 sec. Since reduced phonation time is a general sign of insufficient glottal closure, it reflects the degree of glottal deficiency also in this type of paralysis.

3. Lowered speaking pitch. It is very characteristic that these patients complain of a marked lowering of the speaking voice. This is most embarrassing to women who may find themselves talking in the male range. Once the other possible causes of this symptom, such as hypertrophic laryngitis, hormonal masculinization, or psychogenic ventricular dysphonia, are ruled out, the diagnosis of cricothyroid paralysis is almost established by this vocal symptom. Most of the described cases were found to speak in a low male range. This means that the female speaking pitch is lowered to a greater extent than in males. Undoubtedly, this descent of the speaking level reflects the loss of vocal cord tension.

4. Reduction of vocal range is equally characteristic. Usually, the residual range is reduced to 5-10 semitones in the chest register. It follows that all higher tones in the middle and high registers are lost as a result of absent vocal cord tension. Looking back at our Figures 14-16, we are reminded of the normal finding that cricothyroid activity is minimal in the chest register. Apparently this muscle is not needed for the low tones which explains their preservation after its paralysis.

5. Loss of singing voice and its replacement by great *vocal monotony* are a direct result of the reduced vocal range. For the same reason, previous singers can no longer carry a tune or imitate tonal intervals. All this is due to the loss of vocal cord tension.

DIAGNOSTIC PROCEDURES.

Precise assessment of cricothyroid function is not difficult when the described techniques are employed and evaluated as explained above. These diagnostic procedures are important for two conditions: *1.* Diagnosis of isolated paralysis of the superior laryngeal nerve or its end organ, the cricothyroid

muscle; *2.* evaluation of the influence of the cricothyroid on vocal cord position in paralysis of the inferior laryngeal nerve, particularly in case of bilateral median immobilization. Summarizing the discussion of diagnostic signs, it can be stated that the following findings are important.

1. Diagnosis of Cricothyroid Paralysis.

Laryngoscopic findings: a. Laryngeal anesthesia if both branches of the superior laryngeal nerve are affected; *b.* Deviation of the posterior commissure to the side of paralysis; *c.* Loss of tension in the affected cord which appears flaccid, shortened, and flopping; *d.* Level differences of the cords, the affected cord hanging down with inspiration and bulging upwards in expiration; *e.* Possible hyperemia of the affected cord; and *f.* Possible prolapse of the arytenoid on the side of paralysis.

Vocal changes: a. Weakness, roughness, and great fatigability of the voice; *b.* Shortened phonation time; *c.* Lowered speaking pitch; *d.* Reduction of vocal range to the low tones of chest register; and *e.* Loss of singing voice.

Stroboscopic findings. This well established technique,[49,63,68,74] reveals fine details of vocal cord vibration and demonstrates the reduction of vibration on the affected side. According to previous studies,[2,10,43] the following abnormalities of vocal cord vibration may be seen: *a.* Arrhythmia or disturbed symmetry between the two cords; *b.* Rapid loss of vibrating regularity and appearance of an irregular flutter as a sign of different tension in the two cords; or *c.* Replacement of normal vibrations by pathologic vertical movements of the affected cord, indicating its flaccidity and weakness.

Radiologic findings: a. Absence of progressive narrowing of crico-thyroid space on lateral projection with increasing pitch; *b.* Absence of differences in vocal cord configuration between low and high tones on frontal tomogram; *c.* Absence of vocal cord elongation for high tones on lateral radiographs of younger persons without advanced laryngeal ossification.

Manipulation test: It is well known that various types of

pressure exerted on the thyroid cartilage alter the pitch of simultaneous phonation. *Frontal* pressure on the thyroid prominence (Gutzmann's pressure test) normally lowers the pitch of high tones because it counteracts cricothyroid contraction.[49] *Bilateral* pressure on the thyroid wings normally raises pitch because it tends to lengthen the cords.[75] If these tests produce abnormal results, they tend to confirm disturbed cricothyroid function. More impressive is a test of manual compression of the crico-thyroid distance by *pushing the thyroid down* or the cricoid up. In case of cricothyroid paralysis, this test will tend to raise the pitch of low tones.

2. Cricothyroid Action and Vocal Cord Position.

It is now widely accepted that the cricothyroid muscle determines the position of a paralyzed cord. Numerous authors agree that paramedian position represents complete paralysis of the inferior (recurrent) laryngeal nerve, whereas intermediate position results from combined and complete paralysis of both the superior and inferior laryngeal nerves.[3-7,9,16-18,62,71] Apparent exceptions to this rule, such as intermediate cord position with a thoracic lesion, are explained by Hofer's[23] demonstration of retrograde degeneration of the motor nucleus ambiguus in low vagus lesions with secondary loss of all motor and sensory laryngeal innervation via both laryngeal nerves.

In view of the renewed interest in the problem of auxiliary adduction by the cricothyroid muscle,[22] it is worth while to investigate this function in cases of *bilateral midline paralysis.* As explained by several authors,[5,7,9,71] bilateral immobilization of both cords near the midline occurs by means of several mechanisms, one of which is the adductive component of the cricothyroid as long as its nerve supply is intact. This active muscular component of paramedian immobilization can be demonstrated by several tests.

If these tests for cricothyroid function are positive, interruption of the superior laryngeal nerve, or ablation of the cricothyroid muscle, will produce a widening of the glottis.[71] *a. Cocainization of the cords* is followed by widened glottis when the paramedian position is partly due to adductor spasm

from reflex contracture. Both the afferent and efferent reflex arcs are mediated by the superior laryngeal nerve which is then intact. *b. Novocaine injection into the cricothyroid* is followed by widened glottis as long as this muscle and its nerve are functioning. *c.* The *Probe test of arytenoid mobility* determines fixation of the cords by arthritic ankylosis. If this is present, the cricothyroid no longer influences the cord position and its elimination will not widen the glottis.

Impressive evidence for the adducting action of the cricothyroid and its influence on the paramedian position of a paralyzed cord was presented by recent EMG findings.[11] *a. No difference in electric activity* of the cricothyroid on the normal and paralyzed side was found in cases of unilateral *paramedian* position; hence, it is the intact cricothyroid which maintains the paralyzed cord near the midline. *b.* In contrast, the cricothyroid on the paralyzed side revealed *prolonged action potential duration* in cases of unilateral *intermediate* position; consequently, intermediate position is due to combined and complete paralysis of both laryngeal nerves of one side. This confirms the original view of Wagner and Grossmann which is now held by the majority of authors.[5]

THERAPY.

All recent contributions to cricothyroid pathology[2,10,42,43,49] are unanimous in stressing the poor prognosis of the vocal disability resulting from permanent loss of cricothyroid function. Contrary to the excellent results of vocal rehabilitation in cases of simple recurrent nerve paralysis, no amount of physiotherapy, vocal exercises, or psychotherapy will improve the vocal complaints of persons suffering from paralysis of the superior laryngeal nerve. To improve this hopeless situation, two procedures are herewith recommended which were elaborated on dogs and human cadavers. They will be tried as soon as a suitable and interested patient can be found.

1. Thyrohyoido-Cricoido-Raphy.

Procedure of suturing the thyrohyoid muscle to the cricoid. Anesthesia may be general or local. A small horizontal skin

incision is made at the level of the cricothyroid space. The strap muscles are separated in the midline, exposing the thyroid cartilage. The thyrohyoid muscle is then freed from its insertion on the *linea obliqua* and sufficiently mobilized to be pulled down to the cricothyroid muscle. If the latter is fairly well preserved, the thyrohyoid is broadly united with it by strong mattress sutures of heavy chromic catgut.

If the cricothyroid muscle is found atrophic, the thyrohyoid is sutured to the cricoid periosteum. In case this attempt would result in excessive tension, another modification will be preferable. The thyrohyoid is then artificially elongated by elevating the common insertion of the sternothyroid and thyrohyoid muscles at the *linea obliqua* and by cutting out a cranial portion of the sternothyroid muscle. This combined strap of thyrohyoid and sternohyoid muscle will then be long enough to reach the cricoid cartilage where it is sutured to its periosteum. The remaining caudal portion of the sternothyroid may be reunited with the thyroid periosteum near the inferior thyroid margin.

Rationale: Since the thyrohyoid muscle elevates the thyroid, it distends the cricothyroid distance. If, therefore, this muscle is attached to the cricoid, it will elevate the anterior cricoid ring and thus reduce the cricothyroid distance. While substituting for the lost cricothyroid muscle function, it will continue to act as a laryngeal elevator. Actual trial on a patient should readily demonstrate whether this reconstructed muscle action can improve the voice as is to be expected from theoretical consideration. No untoward results should ensue from this relatively minor surgical procedure.

2. Crico-thyroido-pexy.

Should a patient be disinclined to undergo this harmless myoplastic operation, a simpler procedure may be suggested. Although this method is less physiologic than the first one and does not establish a flexible mechanism of cricothyroid approximation, it can still improve the main complaint of an excessively low, weak, and fatigued voice.

Procedure of fixing the cricoid ring to the thyroid cartilage.

Anesthesia must be local. After exposure of the cricothyroid space as described above, the cricoid ring is elevated with a hook while the patient phonates a sustained tone. Various levels of elevation are tried until the optimum rise of vocal pitch is heard. This level of cricothyroid approximation is then permanently maintained by tying the two cartilages together with tantalum wire. In case of unilateral paralysis, it may be best to elevate the paralyzed side. If both muscles are affected, elevation must occur in the midline.

For this purpose, a thin hole is bored or drilled through the thyroid cartilage near its inferior margin, laterally or in the midline. A curved needle is led through the hole until it emerges through the cricothyroid membrane below the inferior thyroid margin. The needle is then re-inserted through the cricothyroid membrane just above the cricoid and led around this cartilage until it emerges below its lower margin. Care must be taken not to penetrate the laryngeal mucosa. Finally, the two wire ends are tied over the cricothyroid space with sufficient tension to ensure permanent elevation of the cricoid.

This anterior elevation should reach a level which will maintain appropriate external vocal cord tension for the permanent shift of speaking pitch to a higher level; hence, the patient must be conscious to test his voice throughout the procedure. If the voice becomes too high, the wire tension must be slightly released. The optimal pitch level can be predetermined according to sex, age, body type, and personal preference. Its selection during operation can be objectively controlled with an adjustable tuning fork, pitch whistle, or continuous sweep audiometer.

Rationale: Cricothyroid muscle function is replaced by permanent elevation of the cricoid ring. As a result, external vocal cord tension is maintained at a level which provides acceptable pitch for the speaking voice. In contrast to the myoplastic procedure outlined above, this simple cricoid elevation does not permit major modulations of vocal pitch; therefore, it does not increase the vocal range, because it merely elevates the residual range to a higher pitch level.

CONCLUSIONS.

Although much information on cricothyroid muscle function is available, this knowledge is not used in clinical practice. It was, therefore, felt necessary to present a systematic outline of normal and disturbed cricothyroid function, considering anatomy, nerve supply, physiology, radiologic investigation, electromyographic study, and clinical diagnosis.

It is definitely established that this muscle provides the crude external vocal cord tension of isotonic type which enables the vocalis muscle to exert fine internal cord tension of isometric type. In addition, the cricothyroid is an auxiliary adductor to the paramedian line. Vocal cord tension results in their visible elongation.

Details of cricothyroid action were elaborated by electrical stimulation experiments. Movements of the cricoid cartilage in response to muscular contraction occur in two directions: vertical anterior elevation and horizontal torsion. A disturbance of these movements by unilateral paralysis is responsible for the typical laryngoscopic appearance. The posterior commissure deviates to the side of the paralysis, while the affected cord tends to be lower and shorter.

Basic facts of vocal physiology serve for the precise definition of vocal terminology which is based on accepted acoustic standards. To illustrate the further discussions, the test subject's voice was first recorded on sonagrams. The same specified vocal actions were then investigated with lateral radiography, frontal tomography, and electromyography.

A composite picture emerged which clearly showed that the cricothyroid muscle is the main determinator of higher vocal pitch in the upper two registers. On the basis of present knowledge it appears that cricothyroid activity begins with the transition into the middle register and increases to a maximum with the falsetto register. All findings and conclusions from this study are interpreted in accordance with the classical myo-elastic or aero-dynamic theory of passive vocal vibration.

Further details of interaction between the cricothyroid and

other muscles, such as the sternothyroid, are revealed by electromyography. Several open questions deserve additional study on a larger number of subjects with different vocal types. Such experiments will promote the understanding of laryngeal physiology and help to differentiate the constant general trends of laryngeal movements from the variable individual habits of single persons.

Clinical findings in cricothyroid paralysis can be described with considerable accuracy. It should be generally known that the serrated or serpentine cords shown in many textbooks do not occur in reality. Rather, this paralytic syndrome consists of the following findings: laryngeal anesthesia, if both branches of the superior laryngeal nerve are affected; deviation of the posterior commissure to the side of the lesion; loss of tension in the affected cord with level differences, shortened appearance, and possible hyperemia. Vocal changes, stroboscopic findings, radiologic signs, and results of manipulation tests are equally characteristic.

All these examinations permit the accurate assessment of cricothyroid function. These determinations are important not only for the prompt recognition of isolated cricothyroid paralysis, but also for the management of bilateral laryngeal paralysis with paramedian immobilization. A number of these patients profit from cricothyroid elimination, alone or in conjunction with laterofixation of one cord.

Since no treatment is presently available for the restoration of function after permanent loss of cricothyroid activity, two new surgical procedures are proposed for the improvement of vocal cord tension.

SUMMARY.

The significance of the cricothyroid muscle for human phonation is analyzed from the various viewpoints of its anatomy, physiology, and pathology. Available information is supplemented by new investigations with sonagraphic, radiologic, and electromyographic techniques. Personal observations are compared with previous descriptions of pathologic

findings for the purpose of describing the general symptomatology of cricothyroid paralysis. A proposal of two new surgical procedures for the restoration of vocal cord tension after permanent loss of cricothyroid function may improve the presently poor prognosis of this paralytic syndrome.

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BIBLIOGRAPHY.

1. Amersbach, K.: Elektrophysiologische Untersuchungen an der Kehlkopfmuskulatur. *Ztschr. Ges. Exper. Med.*, 28:122, 1922.

2. Arnold, G. E.: Die Traumatischen und Konstitutionellen Stoerungen der Stimme und Sprache. Wien: Urban und Schwarzenberg, 1948.

3. Arnold, G. E.: Vocal Rehabilitation of Paralytic Dysphonia: I. Cartilage Injection Into a Paralyzed Vocal Cord. *Arch. Otolaryngol.*, 62:1, 1955.

4. Arnold, G. E.: Vocal Rehabilitation of Paralytic Dysphonia: II. Acoustic Analysis of Vocal Function. *Arch. Otolaryngol.*, 62:593, 1955.

5. Arnold, G. E.: Vocal Rehabilitation of Paralytic Dysphonia: III. Present Concepts of Laryngeal Paralysis. *Arch. Otolaryngol.*, 65:317, 1957.

6. Arnold, G. E.: Vocal Rehabilitation of Paralytic Dysphonia: IV. Paralytic Dysphonia Due to Unilateral Recurrent Nerve Paralysis. *Arch. Otolaryngol.*, 68:284, 1958.

7. Arnold, G. E.: Vocal Rehabilitation of Paralytic Dysphonia: V. Vocal Symptomatology After Bilateral Loss of Abduction. *Arch. Otolaryngol.*, 70:444-453, 1959.

8. Bellussi, G., and Visendaz, A.: Il Problema dei Registri Vocali alla Luce della Tecnica Roentgenstratigrafica. *Archivio Ital. Otol. etc.*, 60:130, 1949.

9. Berendes, J.: Neuere Ergebnisse ueber Bewegungsstoerungen des Kehlkopfes. *Archiv Ohr- usw. Heilk*, 169:1-172, 1956.

10. Doehne, E.: Beobachtungen ueber eine Periphere Laehmung des Nervus Laryngeus Superior. *Archiv Sprach-Stimmheilk*, 5:157, 1941.

11. Faaborg-Andersen, K.: Electromyographic Investigation of Intrinsic Laryngeal Muscles in Humans. *Acta Physiol. Scand.*, 41: Supp. 140, 1957.

12. Faaborg-Andersen, K., and Buchthal, F.: Action Potentials from Internal Laryngeal Muscles During Phonation. *Nature*, 177:340, 1956.

12a. Faaborg-Andersen, K., and Sonninen, A.: The Function of the Extrinsic Laryngeal Muscles at Different Pitch. *Acta Otolaryngol.*, 51:89, 1960.

13. Feinstein, B.: The Application of Electromyography to Affections of the Facial and the Intrinsic Laryngeal Muscles. *Proc. Roy. Soc. Med.*, 39:817, 1945-46.

14. Fink, B. R., Basek, M., and Epanchin, V.: The Mechanism of Opening of the Human Larynx. The Laryngoscope, 66:410, 1956.

15. Fischer, J. F., and Moeller, J.: Beitraege zur Kenntnis des Mechanismus der Brust- und Falsettstimme. *Monatsschr. f. Ohrenh.*, 42:8, 1908.

16. Fischer, N. D.: Preliminary Report on an Application of the Motor Function of the Superior Laryngeal Nerve. *Ann. Otol., Rhinol. and Laryngol.*, 61:352, 1952.

17. Freedman, L. M.: The Role of the Cricothyroid Muscle in Tension of the Vocal Cords. *Arch. Otolaryngol.*, 62:347, 1955.

18. Freedman, L. M.: The Role of the Cricothyroid Muscle in Tension of the Vocal Cords. The Laryngoscope, 66:574, 1956.

19. Garde, E. J.: Observation Stroboscopique de la Vibration des Cordes Vocales dans le "Petit Registre" (ou Registre "de Sifflet") des Soprani Suraigus. *Folia Phoniat.*, 3:248, 1951.

20. Gregg, R.: Experimental Laryngeal Paralysis. *Ann. Otol., Rhinol. and Laryngol.*, 65:639, 1956.

21. Gregg, R. L.: Avoiding Injury to the Extralaryngeal Nerves. *Ann. Otol., Rhinol. and Laryngol.*, 66:656, 1957.

22. Grossmann, M.: Ueber den M. Cricothyreoideus. *Monatsschr. f. Ohrenh.*, 34:177, 1900.

23. Hofer, G.: Zur Motorischen Innervation des Menschlichen Kehlkopfes. *Z. Ges. Neurol. Psych.*, 177:783, 1944.

23a. Hollien, H., and Moore, G. P.: Measurements of the Vocal Folds During Changes in Pitch. *Jour. Speech Hearing Res.*, 3:157, 1960.

24. Hooper, F. H.: Experimental Researches on the Tension of the Vocal Bands. *Arch. Laryngol.*, 4 (supp.): 1, 1883.

25. Isshiki, N.: Regulatory Mechanism of the Pitch and Volume of Voice. *Oto-Rhino-Laryngol. Clinic Kyoto*, 52:1065, 1959.

26. Jeschek, J.: Ueber Wechselseitige Funktionsbeziehungen der Kehlkopfmuskulatur. *Archiv Ohren- usw. Heilk.*, 163:372, 1953.

27. Jeschek, J.: Ueber den Derzeitigen Stand der Forschung auf dem Gebiet der Stimmbandlaehmungen. *Folia Phoniat.*, 10:129, 1958.

28. Jeschek, J.: Die Ektomie des M. Cricothyreoideus als Zusaetzlicher Eingriff zur Laterofixation. *Archiv Ohren- usw. Heilk.*, 173:433, 1958.

29. Judson, L. S., and Weaver, A. T.: Voice Science. New York: Appleton-Century-Crofts, 1942.

30. Jurasz, A.: Zur Frage nach der Wirkung der Mm. Thyreocricoidei. *Archiv Laryngol.*, 12:61, 1901.

31. Kaiser, L.: Manual of Phonetics. Amsterdam: North-Holland Publishing Company, 1957.

32. Kaspar, F.: Zur Technik der Kropfoperation. *Deutsche Z. Chir.*, 256:4, 1942.

33. Katzenstein, J.: Ueber die Innervation des M. Crico-Thyreoideus. *Virchow's Archiv*, 130:316, 1892.

34. Katzenstein, J.: Weitere Mittheilungen Ueber die Innervation des M. Crico-Thyreoideus. *Virchow's Archiv*, 136:203, 1894.

35. Katzenstein, J.: Ueber Probleme und Fortschritte in der Erkenntnis der Vorgaenge bei der Menschlichen Lautgebung etc. Passow-Schaefer Beitrage, 3:291, 1909.

36. KATZENSTEIN, J.: Ueber Brust-, Mittel- und Falsettstimme. Passow-Schaefer Beitrage, 4:271, 1911.

37. KECHT, B.: Die Behandlung der Operativen Rekurrenslaehmungen. Wien: Maudrich, 1953.

38. KRAUSE, H.: Ueber den Musculus Cricothyreoideus. *Monatsschr. f. Ohrenh.*, 35:2, 1901.

39. KUTTNER, A., and KATZENSTEIN, J.: Bemerkung zu der M. Grossmann'schen Arbeit "Ueber den M. Cricothyreoideus." *Monatsschr. f. Ohrenh.*, 34:7, 1900.

40. LAWSON, L. J.: The Superior and Recurrent Laryngeal Nerves. *Quar. Bull. Northwest. Univ. Med. School*, 22:356, 1948.

41. LONGET: Recherches Expérimentales sur les Nerfs des Muscles du Larynx, etc. *Gaz. Med. Paris*, 1841.

42. LUCHSINGER, R.: Ueber die Periphere Isolierte Laehmung des Nervus Laryngeus Superior. *Archiv Sprach- Stimmheilk.*, 4:15, 1940.

43. LUCHSINGER, R.: Die Periphere Isolierte Laehmung des N. Laryngeus Superior. *Archiv Ohren- usw. Heilk.*, 151:393, 1942.

44. LUCHSINGER, R.: Kasuistischer Beitrag zu den Kehlkopflaehmungen bei Lues II. *Schweiz. Med. Woschr.*, 72:477, 1942.

45. LUCHSINGER, R.: Stimmstoerung nach Struma-Operation ohne Rekurrensschaedigung. *Schweiz. Med. Woschr.*, 72:1136, 1942.

46. LUCHSINGER, R.: Die Zentrale Laehmung des N. Laryngeus Superior. *Pract. Oto-Rhino-Laryngol.*, 7:139, 1945.

47. LUCHSINGER, R.: Falsett und Vollton der Kopfstimme. *Archiv Ohren- usw. Heilk.*, 155:505, 1949.

48. LUCHSINGER, R.: Zur Kenntnis des Mechanismus der Brust- und Kopfstimme. *Pract. Oto-Laryngol.*, 12:311, 1950.

49. LUCHSINGER, R., and ARNOLD, G. E.: Lehrbuch der Stimm- und Sprachheilkunde. Ed. 2, Wien: Springer-Verlag, 1959.

50. MAINZER, F. S.: Paralysis of the Superior Laryngeal Nerve as a Result of Cerebral Hemorrhage. THE LARYNGOSCOPE, 41:33, 1931.

51. MASSEI, F.: Ueber die Bedeutung der "Anaesthesie des Kehlkopfeingangs" bei den Recurrenslaehmungen. *Berl. Klin. Woschr.*, Vol. 47, 1906.

52. MAYET, A.: Zur Innervation des M. Cricothyreoideus. *Anat. Anz.*, 103:340, 1956.

53. MAYET, A., and MUENDNICH, K.: Beitrag zur Anatomie und zur Funktion des M. Cricothyreoideus und der Cricothyreoidgelenke. *Acta Anatomica*, 33:273, 1958.

54. MOELLER, J., and FISCHER, J. F.: Ueber die Wirkung des M. Cricothyreoideus and Thyreoarytaenoideus Internus. *Archiv Laryngol.*, 15:72, 1904.

55. MORAN, R. E., and CASTRO, A. F.: The Superior Laryngeal Nerve in Thyroid Surgery. *Ann. Surg.*, 134:1018-1021, 1951.

56. MUENDNICH, K.: Anatomische und Histologische Untersuchungen und Experimente zur Physiologie und Pathologie des Menschlichen Kehlkopfes. *Archiv Ohren- usw. Heilk.*, 169:190, 1956.

56a. MURTAGH, J. A., and CAMPBELL, C. J.: The Effect of the Barbiturates on Laryngeal Function. *Ann. Otol., Rhinol. and Laryngol.*, 65:545, 1956.

57. Mygind, H.: Die Paralyse des M. Cricothyreoideus. *Archiv Laryngol.*, 18:403, 1906.

58. Némai, J.: Zur Anatomie und Physiologie des Stimmorgans. *Monatsschr. f. Ohrenh.*, 71:74, 1937.

59. Neumayer, H.: Untersuchungen Ueber die Funktion der Kehlkopfmuskeln. *Archiv Laryngol.*, 4:323, 1896.

60. Pernkopf, E.: Topographische Anatomie, Vol. III. Wien: Urban und Schwarzenberg, 1952.

61. Pressman, J. J.: Physiology of the Vocal Cords in Phonation and Respiration. *Arch. Otolaryngol.*, 35:355-398, 1942.

62. Pressman, J. J., and Kelemen, G.: Physiology of the Larynx. *Physiol. Reviews*, 35:506, 1955.

63. Ranke, O. F., and Lullies, H.: Gehoer, Stimme, Sprache. Berlin: Springer-Verlag, 1953.

64. Russell, G. O.: Down-Pulling of the Thyroid on the Cricoid May Lower Voice Pitch. *Arch. Néerland. Phonét. Expér.*, 6:88, 1931.

65. Sokolowsky, R.: Analytisches zur Registerfrage. Passow-Schaefer Beitrage, 6:75, 1912.

66. Sonninen, A.: Is the Length of the Vocal Cords the Same at All Different Levels of Singing? *Acta Otolaryngol.*, Suppl. 118:219, 1954.

67. Stupka, W.: Experimentelle Beitraege zur Kenntnis der Atembewegungen des Hundekehlkopfes. *Z. Hals- usw. Heilk.*, 9:306, 1924.

68. Tarneaud, J.: La Stroboscopie du Larynx. Paris: Maloine, 1937.

69. Tarneaud, J.: Die Stimmlippe im Zustand der Phonation. *Hals- usw. Arzt*, 28:36, 1937.

70. Tschiassny, K.: Studies Concerning the Action of the Musculus Cricothyreoideus. The Laryngoscope, 54:589, 1944.

71. Tschiassny, K.: Therapeutically Induced Paralysis of the Cricothyroid Muscle or Its Removal in Paralytic Laryngeal Stenosis. *Arch. Otolaryngol.*, 65:133-142, 1957.

72. Weleminsky, J.: Zur Physiologie und Pathologie der Bewegungsvorgaenge am Kehlkopf. *Z. Hals- usw. Heilk.*, 16:374, 1926.

73. Wenrick, J. E.: Stimulation of Muscles in the Larynx Tilting the Thyroid Downward on the Cricoid Lowers Pitch. *Arch. Neérland. Phonét. Expér.*, 6:92, 1931.

74. Zaifol, H.: Utilisation de la Stroboscopie en Clinique Laryngologique et Phoniatrique. Paris: Foulon, 1941.

75. Zenker, W., and Zenker, A.: Ueber die Regelung der Stimmlippenspannung Durch von Aussen Eingreifende Mechanismen. *Folia Phoniat.*, 12:1-36, 1960.

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TWENTY YEARS' EXPERIENCE WITH LABYRINTH SURGERY FOR MENIERE'S DISEASE.*

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The impetus which started this work was caused by two events: first was my experience with a case which I saw in consultation, in which there was an active fistula into the labyrinth from an unhealed radical mastoidectomy, causing repeated attacks of vertigo. The patient was desperate and threatened suicide, and I advised a labyrinthectomy, in spite of the risks involved. This was before the antibiotic era. With the idea of trying to seal off the meninges and prevent a meningitis, I used a coagulating current in the labyrinth. The patient made an uneventful convalescence but astonished everyone concerned by the fact that the hearing in the operated ear remained at the preoperative level. The second event was the publication by Hallpike and Cairns in 1938, of the pathologic findings of a case of Meniere's disease, showing for the first time that a severe hydrops or distention of the membranous labyrinth was present.

Having seen several patients who had undergone an VIIIth nerve section for this condition, with rather poor results, I began to wonder whether direct attack on the labyrinth itself

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would not prove to be a better method of combating this ailment, especially if hearing could be preserved. With the blessing of George Wright, who was professor of neurology at the University of Pittsburgh at that time, I began to operate on the labyrinth for Meniere's disease. The procedure which I employed was to open the mastoid antrum by way of a postauricular approach and to remove the outer wall of the aditus until the incus was exposed. The ampulla of the horizontal semicircular canal was opened with a drill, then a fine neurosurgical needle electrode was passed forward into the vestibule, and an attempt made to destroy the utricle and semicircular canals without destroying the saccule, in the hope of preserving hearing. The results were completely unpredictable. In a series of 53 operations, hearing was preserved in 15 and completely lost in 38. The 38 cases in which hearing was destroyed, however, had complete relief of symptoms.

Of the 15 cases in which hearing was preserved only four had complete relief of symptoms with hearing maintained at the preoperative level or above for a period of five years. This is less than 8 per cent. The hearing of the remaining 11 cases continued to deteriorate. Seven of these patients complained bitterly of roaring tinnitus and distortion of hearing, and four of them continued to have vertiginous symptoms, apparently coming from the posterior semicircular canal. Revisions were done on five of these, with labyrinthectomies and subsequent complete relief.

As I saw more and more cases of Meniere's disease and, with a better understanding and insight into the true nature of the condition, I abandoned the attempt to preserve hearing. In 1950 before the American Otological Society, in this city, I gave a detailed report of the symptomatology of Meniere's disease for the first time, with the conclusion that it is primarily a cochlear disturbance and that attacks of vertigo occurred either concomitantly with the onset of hearing disturbance, or later. An analysis of 220 cases of Meniere's disease at that time revealed about 10 per cent to be bilateral; however, most of these cases were bilateral from the time

of onset of symptoms. Only four cases developed the disease in the unaffected ear more than six months after involvement of the first ear. Consequently, it seemed illogical to attempt to preserve distorted hearing in one ear for fear of later involvement of the other ear. In the early years of this work I operated upon one ear of two bilateral cases with poor results. The hearing was lost in the operated ear, and attacks of vertigo continued along with decreasing hearing in the second ear.

I believe in our present state of knowledge that all bilateral cases must be handled as a medical problem. In my report in 1950 I advocated complete destruction of the involved labyrinth in unilateral cases with no attempt to preserve hearing. I still hold that opinion.

My original surgical procedure fell into some disrepute because other men attempting it reported a number of facial paralyses; some temporary and some permanent. This was due to the improper use of the needle electrode. If it should come in contact with the lateral wall of the labyrinth some facial damage is bound to occur. I no longer advise its employment, although I still use it after extracting the membranous labyrinth, because I believe that coagulating the raw nerve endings insures a smoother and more rapid postoperative convalescence. This is a matter of personal preference. I can report that, up to the present time, no patient upon whom I have operated by this method has ever shown any sign of facial nerve damage; in fact, in 144 cases operated upon by Jordan and myself to date, the mortality and morbidity have been nil.

In this series of operated cases, I believe that the correct diagnosis was made in all but one. This case was referred to me from the neurological department of the hospital for surgery for Meniere's disease. There was no demonstrable hearing in the affected ear which should have made me suspicious. Three months later this case developed cerebellar signs of incoordination and loss of corneal reflexes. A craniotomy revealed a large meningioma.

What about the subsequent course of our operated cases?

How many showed delayed involvement of the unoperated ear? To date, three cases have shown signs of Meniere's disease in the unoperated ear. One case showed only a cochlear involvement. With prompt and early treatment the symptoms subsided, and normal hearing has been maintained.

In the second case active therapy has been employed for five years and usable hearing is being maintained in the unoperated ear. The third case is most interesting. The second ear became involved 11 years following operation. Fortunately for this patient, he was one of the four who maintained hearing in the operated ear at or above the preoperative level. He now wears a hearing aid in the operated ear with a discrimination score of 56 per cent; the same as before operation. The hearing in the unoperated ear has gradually deteriorated and shows a discrimination score of only 12 per cent.

I do not wish to give the impression that I am an eager beaver for surgery for Meniere's disease. Over the past 20 years we have controlled 88 per cent of our cases with medical therapy, more or less satisfactorily, and have operated upon only 12 per cent of 1200 cases.

In the past few years some new types of surgical treatment have been employed for this condition. A very satisfactory procedure is to operate by way of the external auditory meatus, lift up the drum, cut the stapedius tendon and lift the stapes from the oval window while maintaining its attachment to the incus; then, with dental broach, picks and suction, the contents of the labyrinth can be removed and the stapes replaced in the oval window. This procedure actually can be done under local anesthesia with little discomfort to the patient. The important thing to note is, that at the conclusion of the operative procedure, any opening into the labyrinth should be closed or sealed off, whether by means of the stapes in the oval window or bone mush in the dome of the vestibule, so that a subsequent upper respiratory or middle ear infection cannot travel to the meninges by way of the labyrinth.

The main purpose of this paper is to decry the use of

ultrasonic sound in an attempt to destroy the vestibular labyrinth and preserve hearing. The procedure itself is a very tedious one. The ultrasonic wave applicator is not standardized and is very difficult to calibrate accurately. A simple mastoidectomy must be done first to provide room for the applicator which is applied to the dome of the vestibule for as long as 30 to 40 minutes. The results of ultrasonic therapy are as completely unpredictable as were the results of my original procedure with the coagulating needle electrode. The ampullae of the horizontal and superior canals can be destroyed satisfactorily with this procedure, but the ampulla of the posterior semicircular canal lies too deeply to be reached from the dome of the vestibule without also damaging the cochlea and endangering the facial nerve. I have discovered, to my sorrow, that by preserving the cochlear function we actually are doing our patients a disservice. The preservation of the cochlear symptoms of Meniere's disease results in much distress, and these symptoms can be nearly as incapacitating as the attacks of vertigo.

I have had no personal experience with the use of the ultrasonic applicator, but I have seen two patients who submitted to this therapy: one sustained a facial paralysis with subsequent partial recovery. The other patient I shall report in more detail. He was an experienced radio and television announcer with excellent hopes for advancement in his profession. Due to frequent attacks of vertigo and enforced absence from his job, he failed to obtain expected promotions. Eighteen months ago he underwent ultrasonic therapy to his affected labyrinth with no improvement of his symptoms; in fact, he developed a tendency to fall over backwards whenever he had an attack of vertigo. The distortion of sound and roaring tinnitus made it difficult for him to enunciate clearly when broadcasting. He reported that sounds coming through his headphones set him half crazy. He had developed a neurodermatitis of his face which prevented his appearing on television and he was in constant fear of losing his job. Four months ago I operated upon his affected labyrinth and destroyed it with gratifying results. Not only has he been enabled to resume his job but the neurodermatitis has dis-

appeared. He is again appearing on television and recently signed a lucrative commercial contract.

To summarize my views on Meniere's disease:

1. It is a definite clinical entity showing symptoms and signs which sharply delineate it. It is primarily a cochlear disturbance and the diagnosis can and should be made from the cochlear findings alone. If this is done and prompt treatment undertaken, vestibular disturbances may be avoided. It is no longer justifiable to label a case as having Meniere's disease because it presents the triad of symptoms of deafness, tinnitus and vertigo, called Meniere's syndrome.

2. There is no specific medical therapy known to date which can be called a cure for this malady.

3. There are various remedies which will control the active manifestations of Meniere's disease in the majority of cases, and many cases will have remissions without any therapy.

4. The value of any therapy employed may be partly due to the psychologic relief of the patient in the belief that something is being done for his ailment, resulting in the lessening of emotional and nervous tension.

5. Intractable cases of unilateral Meniere's disease which fail to respond to conservative therapy can be cured by destruction of the involved labyrinth.

6. When there is a serious economic problem and a patient is losing much time from work because of his ailment, surgery should be considered.

7. When an occupational hazard is involved and there is danger of actual injury to the patient when he has an attack, surgery is the treatment of choice. This is especially true of truck drivers and construction workers.

8. Differential surgery of the labyrinth and attempts to preserve the cochlear function are not justifed at present because:

a. the results are completely unpredictable;

b. the preservation of an affected cochlea results in the preservation and continuation of distressing hearing disturbances;

c. when cochlear function is preserved there is a likelihood of an incomplete destruction of the vestibular mechanism and vertiginous symptoms may continue.

9. Labyrinthectomy is uniformly successful in terminating the symptoms of unilateral Meniere's disease.

10. Delayed involvement of the unaffected ear is rare. In our series it was less than 2 per cent of over 1200 cases.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The section of Ophthalmology and Otolaryngology of the Southern Medical Association announces that it is now accepting papers by physicians of either specialty, living in the area of the Southern Medical Association, for consideration for presentation at the next annual meeting to be held in Dallas, Texas, from November 6 to 9, 1961.

The paper or an abstract of the paper may be sent directly to the Secretary, Dr. Albert C. Esposito, Suite 1212, First Huntington National Bank Building, Huntington, West Virginia, as soon as possible.

BELL'S PALSY: A MUCH MISUSED DIAGNOSIS.*†

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The patient who develops a facial paralysis, without obvious cause, such as trauma or otitis media, seldom initially consults an otolaryngologist. The physician first consulted is either the general practitioner, a neurologist or an internist. It would be most unusual, in my opinion, that the physician so consulted would be capable of examining the ears of his patients adequately. This is not surprising considering the inadequate time devoted to otolaryngology in the curricula of most medical schools.

Lacking a history of trauma or of draining ear, a diagnosis of Bell's palsy is made, and the patient is given cortico steroids and told that he has perhaps 80 to 90 per cent chance that his face will start to move again soon and, in all probability, will recover completely. I think both the advice and the treatment are in error. Apropos the use of cortico steroids in Bell's palsy, there is a most informative article by Derrick Travener of England (Lancet. 1954, II:1052) who used Cortisone in a series of patients with Bell's palsy and observed similar patients with no treatment. He reports no difference either in the extent or in the time of recovery with the use of Cortisone. It has been my experience that when the patient with the Bell's palsy starts to recover fairly early the recovery will, in all probability, be complete; but where the start of recovery is delayed it is seldom, if ever, complete. In the event that the paralysis does not clear completely, the patient may be referred to an otologist three to six months or longer after the onset of his paralysis—too late, of course, to be really useful.

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In the past few years I have observed several cases which illustrate the point I am trying to make, which is that every case diagnosed as a Bell's palsy should be seen by a competent otologist. My reasons for this statement are as follows:

If, indeed, the patient is suffering from idiopathic facial paralysis (Bell's palsy), some consideration should be given early to surgical decompression of the nerve, and the otologist should make the decision as to when this interference should be accomplished. It is my opinion that although a high percentage of patients with Bell's palsy recovers spontaneously, complete return of function occurs only in those cases which show evidence of return within a short time. In cases in which there is no sign of return of function in four to six weeks, and in which a complete reaction of degeneration is found, when function does return it is never complete. I am in complete agreement with Dr. Martin when he says that it would be better in all cases to do nothing rather than to have a decompression done by a surgeon unfamiliar with the temporal bone. If, however, the otologist is competent and has respect for soft tissue, I believe decompression should be considered at approximately one month or even less. In any event, the decision to interfere should be the otologist's and careful examination of the ear, with hearing and vestibular studies, is indicated.

Lack of a careful otological examination has caused a permanent irreparable disfigurement which could have been avoided in some cases which I have seen and will describe.

Case 1. In September, 1950, Mrs. B. R., a 46-year-old lady, was sent to me by a neurologist for the express purpose of having her right facial nerve decompressed. The history was that she had been seen by this neurologist seven years previously, complaining of a complete right facial paralysis. A diagnosis of Bell's palsy was made, and she was told that nothing could be done about it. There had been no recovery of function, and when I saw her she had a complete atrophy of the muscles of the right side of her face. Examination of the ear however, revealed a mass in the middle ear which caused a bulge in the posterior half of the drum. This mass was firm and of the consistency of rubber. I made a diagnosis of neurinoma of the facial nerve and operated upon her in April, 1951, and removed a neurinoma of the VIIth nerve. It extended from perhaps 2-3 mm. above the stylo mastoid foramen to just below the geniculate ganglion. I believe this patient's disability could have been avoided had one been permitted to examine her at the onset of the paralysis.

Case 2. Mr. G. A. L., age 44, was seen in May, 1958, in consultation

with Dr. Leonard Furlow (a neurosurgeon). He had served in the Navy during World War II and was exposed to the noise of 5-inch guns and occasionally was temporarily deafened by this noise. In 1946, after discharge from the service, he gradually developed a weakness of the right side of his face and in 1947 went to the Mayo Clinic. He had a right facial paresis with decreased taste on the right anterior portion of his tongue, decreased hearing in the right ear and no other abnormal neurological findings. X-rays of his skull were negative. His condition was unchanged until 1950 when he was struck over the right side of his face with a baseball bat. His jaw was fractured. The cornea was lacerated, and this produced permanent visual impairment. In 1953 he had several dizzy spells and again returned to the Mayo Clinic. Apparently there had been no progression and a diagnosis of an angle lesion could not be made, so he was sent home again. There was no recurrence of dizziness, and he continued to work regularly until nine days prior to admission when he had a nocturnal convulsion with incontinence. This was followed by a short period of combative behavior, and he was hospitalized. Spinal puncture showed increased pressures and protein, so he was transferred to Barnes Hospital. Neurological examination showed a dilated and irregular right pupil with a corneal scar, a complete long-standing peripheral facial paralysis on the right side and a deaf right ear. Audiometry showed a completely deaf right ear, but caloric tests showed reaction from both vestibules. A mass could be seen in the middle ear bulging against the posterior edge of the tympanic membrane which moved freely over it. Spinal puncture showed clear fluid with pressure of 190 mms. of water and a total protein of 143 mgms. per cent.

This man had a VIIth nerve tumor which had eroded through the bone and extended into the middle cranial fossa. Craniotomy was done, and the large smooth tumor, easily separated from the brain, was removed from the middle fossa. It was yellowish in color and contained a few cysts. It was firmly attached to about the mid portion of the petrous portion of the temporal bone. The dura was split around the point of attachment, and some adjacent dura, which was invaded by tumor, was removed. Additional tumor was curetted out of a cavity which was thought to be the middle ear. For radiographic identification, a clip was placed in the bone opening and the wound closed. Approximately six weeks after this operation the mastoid was opened using an endaural approach, and, in the antrum, tumor could be seen extending from the middle ear. Tumor filled the middle ear, and no remnant of facial nerve could be seen. Just anterior to the usual location of the geniculate ganglion, a large hole in the bone contained tumor. When this tumor was removed, the silver clip previously placed there was found to be attached to it. All of the tumor was removed from the middle ear and the mastoid. Oxycel was packed into the dehiscence in the middle fossa, and the rest of the cavity was packed with iodoform gauze. This patient eventually made complete recovery. It was interesting that the neurinoma of the facial nerve in the middle ear had been completely missed in the earlier examinations. The tumor had grown up through the tegmen into the middle cranial fossa.

Case 3, which is of interest, is that of Miss J. B., 16 years old, who was seen first in October, 1957, with a complete left facial paralysis. The story was that she had developed a facial paralysis three years previously for no apparent reason. It was diagnosed as Bell's palsy by a neurologist, and she was treated with electrical stimulation. Her mother told me that this paralysis recovered about 75 per cent, and then recurred on another occasion and was treated with injections of Cortisone with about 40 per cent recovery. Since the previous June the girl had had a complete facial paralysis and had come in to see me on the recommendation of another otolaryngologist who felt that something might be done

about the face. When I examined the girl the left ear drum showed a rather white thick bulging portion anteriorly. I could see the handle of the malleus; but the drum moved very poorly on positive and negative pressure, and I had the impression of a middle ear filled with some firm tissue. I then asked the mother specifically whether the girl had ever had any running ear and was told that she had had an acute otitis media with profuse discharge from the left ear when she was five years of age. A polyp which came through the drum was removed about that time. This infection had subsided on large doses of crystalline penicillin, and she had no further trouble with the ear. There was complete deafness and no caloric response on the left side. X-rays showed a sclerotic mastoid on the left side, and I operated upon this girl with the objective of exploring the middle ear and decompressing the facial nerve. The operation was done November 9, 1957, through a post auricular incision, and there was destruction noted in the tip and in the sino-dural angle. As one went forward through the aditus, cholesteatoma was found filling the attic, and it had eroded the bone over the geniculate ganglion. The geniculate ganglion and some of the facial nerve were exposed by cholesteatoma and the cholesteatoma had to be dissected off it. The nerve was decompressed from the stylo mastoid foramen to the geniculate ganglion, and the sheath was slit. The nerve was completely intact but had been pressed on by cholesteatoma. What amounted to a radical mastoidectomy was done because there was no hearing in this ear at all. Since that time there has been some recovery of function. At rest the girl looks to be in good condition, but cannot close her eye or move her face completely. This degree of recovery took more than a year.

I believe these cases illustrate the importance of a thorough otological examination in patients who have a facial paralysis, and I am confident that in each of these cases, had an otologist examined the patient and taken the appropriate steps early, permanent disfigurement could have been avoided.

These cases and a fourth more tragic one serve to emphasize the fact that all facial paralysis is not Bell's palsy.

Case 4 is that of C. S., age three years. She was sent to me by the same neurologist as B. R. and J. B. There had been present a complete facial paralysis on the left side for six weeks. I was asked to decompress the nerve, the diagnosis of Bell's palsy having been made. I did, indeed, decompress this nerve from the stylo-mastoid foramen to its entrance to the middle ear. When the sheath was slit the nerve did not bulge and looked quite normal. I told the neurologist that this was not Bell's palsy. The child was admitted to Children's Hospital some four months later, in coma, and she died. A large medulloblastoma was found in the angle.

My plea, then, is for careful otologic examination in all cases of facial paralysis, and a further plea that serious consideration be given to surgical interference and decompression when there is no evidence of recovery of function at the end of a month. I believe that it is to the best interest of the patient that consultation between various specialists be freely asked and given in cases of facial paralysis.

THE EFFECT OF ESTROGENS ON MAST CELLS AND ENZYMES IN CONNECTIVE TISSUE METABOLISM IN HUMANS.*†

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INTRODUCTION.

In investigating the effects of drugs and hormones on the tissues of the body it is becoming increasingly apparent that cause and effect can be better related by studying the molecular and enzymatic interrelationships. Since substances from the capillary going to the cell must traverse the ground substance, it is important to study the physical and biochemical behavior of some of its macromolecules and those cells which are presumed to produce them. These affect the life and death of a given tissue. Any substance which alters their status has vital pertinence.

Beyond the realm of the capillary one enters the realm of intercellular and intracellular metabolism which is the impact zone of substances which specifically alter the nutrition of tissues either by changing vessel state or influencing the many cellular enzyme systems involved, or both.

In a clinical study[1] a distinct and convincing increase in the capillary strength was noted following estrogen injection. This was indicated by the altered ability of these capillaries

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to be fractured under the standard stress of a petechiometer. In these cases there was a decrease of over 50 per cent of the number of petechiae within 30 minutes following intravenous injection of Premarin(R). The significance of this improvement in the status of the capillary is of considerable importance, particularly when one speculates on its function in such end organs as the eye or inner ear. If the intracellular metabolism of these end organs were favorably affected, then studies of such influencing factors would be of utmost importance.

From our previous study[2] it was apparent that the water soluble conjugated estrogens (Premarin Intravenous) could affect the ground substance of the body, particularly in the perivascular area and thereby presumably affect capillary strength. During this investigation it was noted that there was a change in the mast cell population and activity after the administration of this estrogen complex. The present study was undertaken to show the effect of surgery and Premarin on the connective tissue with particular reference to mast cell count. A theory is proposed to explain and correlate the role of the mast cell and enzyme function in maintaining the integrity of this mesenchymal tissue.

TECHNIQUE.

An attempt was made to find a biochemical and histochemical correlation of the effect of estrogens on certain tissues of the body. Human oral mucosa biopsies were obtained from seven volunteers just before tonsillectomy surgery and at intervals of one-half, two, four and eight hours after surgery with and without estrogens. Venous blood was drawn from the arm coincidental with each biopsy. Four of these patients were given 20 mg. of Premarin Intravenous immediately after the control biopsy. The remaining three cases had the same surgery, but no Premarin. The tissues were fixed in neutral formalin and embedded in paraffin. They were stained with colloidal iron and toluidine blue, with and without hyaluronidase treatment.[2,3] The blood was prepared to study the effect of Premarin on the alpha, beta and gamma

globulins of the untreated and treated subjects, with regard to possible relationships to serum mucoproteins. Results of these electrophoretic biochemical studies were interesting, but inclusive, since the variation in any one study was often greater than the percentage error intrinsic within the method for measurement. In short, we do not as yet have a satisfactory method for measuring such small quantitative biochemical changes.

Tissue mast cells with their purple metachromatic granules were first noted by Ehrlich in 1877 who named them "Mastzellen," a well fed cell of the connective tissue. While histologic study of these cells continued from that time, little was known regarding their function. Mast cells in human nasal polyps, turbinate mucosa and adenoid tissue were studied by Downey[4] in 1913, and he concluded that the "mast cells of the connective tissue are very labile structures which are constantly being used up in the body . . . and are being constantly differentiated within the connective tissue."

Interest in the functions of the mast cell has grown in the last 20 years, and it is believed that they play an important role in ground substance changes in various physiological states. Mast cells in the pregranular stage are indistinguishable from other cells in the connective tissue. It is probable that they are derived from the primitive mesenchymal cells which are found in the loose connective tissue, especially along the blood vessels. As they change into tissue mast cells and the granules or trephones begin to form, they stain metachromatically with toluidine blue. With the usual hematoxylin and eosin stain, the mast cells are not striking, and it is difficult to study changes as the tissues react to varying stimuli. Colloidal iron gives them a blue color, but neither the granules nor the nucleus is visible.

MICROSCOPIC FINDINGS.

The histochemical studies on the effect of Premarin in humans were of great interest and afforded considerable information. These are in two major categories: *a.* those cases

having surgery and Premarin; *b*. surgery alone without supplemental hormones.

The sections from estrogen treated patients showed responses similar to those in our earlier study. The colloidal iron stain showed an increase in acid mucopolysaccharides for the first four hours and a decrease at eight hours. The total amount of blue-staining mucopolysaccharides at eight hours was still above the level of that shown in the control biopsies. The sections stained with toluidine blue showed a variable increase in metachromasia in the small blood vessel walls and the lumen of the arterioles.

The mucosa from the patients who had not received estrogen showed a very different reaction. The postsurgery sections, stained with colloidal iron, showed no increase in the fine network of blue-staining fibrils of acid mucopolysaccharides. The amount of metachromasia in the sections stained with toluidine blue also showed no change.

The acid mucopolysaccharides, except for chondroitin sulfate B, are depolymerized by hyaluronidase and lose their staining reactions. Mast cell granules, however, are not affected and retain their metachromatic reaction. In order to prevent possible confusion, the mast cells were studied in sections treated with hyaluronidase and stained with toluidine blue (see Fig. 1). The inactive tissue mast cell has a deep reddish purple color which masks the nucleus and the individual granules. As the cell increases in activity, it enlarges, becomes more irregular in shape, the reddish purple granules become discrete, and the pale blue nucleus can be observed. Finally, degranulation of the cell begins, and the granules can be found scattered in the connective tissue. Later by micellophagosis, some of these metachromatic granules appear in the cytoplasm of the fibroblasts where they gradually disappear.

To evaluate the changes, the total number of mast cells and the number of degranulating mast cells were counted. The fibroblasts which had taken up granules were disregarded. Five fields (645×) were counted and the average number calculated for each time interval in each patient.

From these figures, averages for the four patients who had estrogens and surgery, and for the three patients who had surgery alone, were calculated. There were too few cases to make a statistical evaluation absolute, but the differences seem large enough to make them significant.

It will be noted in Table I that the number of mast cells per field in the control groups was comparable. In normal

TABLE I.

Average Number of Mast Cells Per Microscopic Field. (645×)

	Total Mast Cells		Total Degranulating Cells	
Time Interval	Surgery	Premarin & Surgery	Surgery	Premarin & Surgery
Presurgery	5.40	4.65	0.54	0.77
½ Hour Postsurgery	6.40	8.50	0.93	1.44
2 Hour Postsurgery	5.87	10.20	0.87	2.50
4 Hour Postsurgery	4.93	10.50	0.97	2.20
8 Hour Postsurgery	3.70	5.92	0.50	1.12

TABLE II.

Percentage of Change in Average Number of Mast Cells.

	Total Mast Cells		Degranulating Mast Cells	
Time Interval	Surgery	Premarin & Surgery	Surgery	Premarin & Surgery
Presurgery to ½ Hr. Postsurgery	+18.5	+ 82.8	+73.7	+ 87.0
Presurgery to 2 Hr. Postsurgery	+ 8.7	+119.3	+61.1	+225.9
Presurgery to 4 Hr. Postsurgery	- 8.7	+125.8	+79.6	+185.7
Presurgery to 8 Hr. Postsurgery	-31.5	+ 27.3	- 7.4	+ 45.4

connective tissue the number of such cells varies with the species, site, sex and age. No analogous figures for comparison with these human counts were found in the literature.

To obtain more information, the percentage of change (see Table II) in the average number of mast cells from the presurgery baseline to each time level, was calculated. Figure 2 shows that the number of mast cells increased rapidly in the patients receiving estrogens preceding surgery and had not returned to the presurgery level at eight hours. These patients showed a sharp increase in total cell count at each time interval, with a peak of 125.8 per cent at four hours. There

was a prompt drop during the next four hours, and the residual increase over the baseline at eight hours was 27.3 per cent.

In the patients with surgery only, the total mast cells increased slightly in the first half-hour and then decreased, falling below the level of the presurgery count. They showed a moderate increase of 18.5 per cent in the first half-hour, followed by a steady decrease. At eight hours, there was 31.5 per cent less mast cells than in the presurgery controls.

The percentages of degranulating cells in the two groups also showed very different responses. In the estrogen treated patients, the peak of 225.9 per cent was reached at two hours' postsurgery. There was a slight drop at four hours and a sharp drop at eight hours when the residual increase was only 45.5 per cent.

The surgery only patients showed an increase which stayed fairly constant, at approximately 70 per cent, for the first four hours. It then dropped, and at eight hours there was a decrease of 7.4 per cent from the number present in the presurgery controls.

In the estrogen treated patients, there is a close parallel between the amount of acid mucopolysaccharides in the tissue and the changes in the number of mast cells. There was a steady increase in both elements for the first four hours and a decrease at eight hours, although neither had returned to normal at that time.

The sections from the surgery only patients showed no change in the acid mucopolysaccharides during the entire eight hours, while the total number of mast cells showed a slight increase followed by a sharp drop. The relationship between the production of acid mucopolysaccharides and an increase in mast cell activity due to estrogens seems to be established in this study.

It is also interesting to note that the stress of surgery has not seemed to affect the amount of acid mucopolysaccharides but has affected the mast cell activity in spite of a decrease in the total number. The degranulating cells stayed at a more

or less constant level, averaging about 70 per cent above the preoperative baseline for the first four hours and then decreasing sharply to below normal. This would suggest that, while there was little differentiation of primitive mesenchymal cells to mast cells, many of the latter cells present in the tissue had released their granules; thus the changes seen in the endocrine treated cases could not be due to surgery.

Riley[5] believes that mast cells disappear in acute tissue injury. They reappear when new connective tissue fibrils

TABLE III.

Components of Connective Tissue Mast Cells.

1. Heparin[6]	8. Cytochrome Oxidase[11]
2. Histamine[6]	9. Alkaline Phosphatase[11]
3. Serotonin (rats, mice, humans?)[7]	10. Acid Phosphatase[11]
4. Hyaluronic Acid (or precursor)[8]	11. Thermostabile Peroxidase[11]
5. Beta-Glucuronidase[9]	12. Lipase[11]
6. Chymotrypsin-like Enzyme[10]	13. Ribonucleic Acid[12]
7. Phospholipids[11]	

begin to be laid down and when the connective tissue is changing to the gel state. If this is true, it would seem that the estrogens have acted as a protective mechanism in preventing injury from the stress of surgery by strengthening the connective tissue.

One can readily see by the study of this graph (see Fig. 2) that, under the influence of Premarin, the total number of visible mast cells, full of storage granules or trephones, has increased considerably while the number in the untreated group has decreased rapidly. The ratio of degranulating cells to the total mast cells in the treated group is considerably less than a similar ratio in the untreated group.

DISCUSSION.

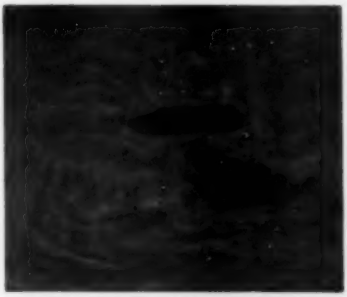
What are the possible explanations for these divergent findings? The insult of surgery produces an increase in degranulation, thereby freeing into the tissue an increased amount of heparin, histamine, serotonin and other substances as noted in Table III.[6,7,8,9,10,11,12] The net effect of these sub-

stances is to induce a dilatation of the small vessels and an increase in capillary permeability. This permits an increase in the fluid content of, and the entrance of, large protein molecules into the ground substance. The clinical manifestation of this is edema, and if gross enough, it would be noted as tissue swelling. Physiologically, however, there is always a certain amount of degranulation taking place, and this is part of the body's homeostatic mechanism whereby substances are brought forth from the capillaries and changes of tissue hydration may be accomplished. These substances are necessary in the tissue and physiopathologically respond to the tissue requirement at that time.

The significance of these findings in the field of general surgery is obvious, but what specific application can such studies have in the field of otolaryngology? In the inner ear labyrinth of thyroidectomized guinea pigs, Poulsen, in his excellent thesis as quoted by Asboe-Hansen,[13] showed that thyrotropin increased the mucopolysaccharide production as well as the number of mast cells. The hexosamine concentration of the endolymph and perilymph did not change, but there appeared to be an increase in the amount of water. In these experiments, he also noted some disturbance of vestibular function.

Some investigators have observed that slow growing tumors often have a considerable number of mast cells in the periphery.[14] In fast growing, highly malignant tumors, this finding is not present. This would suggest that perhaps the desmoplastic attempt which connective tissue makes toward foreign tumor invasion might be related to the mast cell activity in this zone. The role of the mast cell in this regard has not as yet been elucidated.

The effect of hormones on mast cells has been observed by many investigators. Cortisone decreases the number of such cells,[15,16] reduces their activity,[17] and also reduces the content of histamine in connective tissue.[18] Thyrotropin increases the number and activity of the mast cells, loading the cytoplasm with granules.[19,20,21] The increase in the water content of the ground substance may act as a stimulus to



1. Inactive Mast Cells

2. Active Mast Cells

3. Degranulating Mast Cells

4. Fibroblast During Micellophagosis

Fig. 1. The Functional State of the Mast Cell and the Fibroblast. (Toluidine Blue Stain, 900X.)

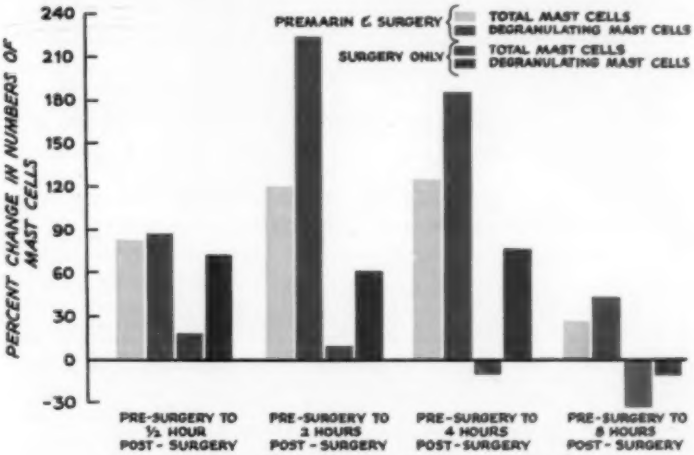


Fig. 2. Effect of Premarin and Surgery on Mast Cells.

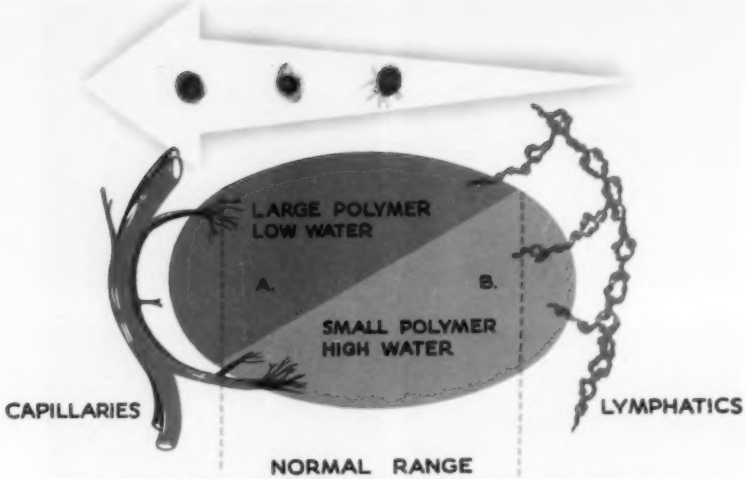


Fig. 3. Effect of Premarin on Ground Substance and Mast Cells.

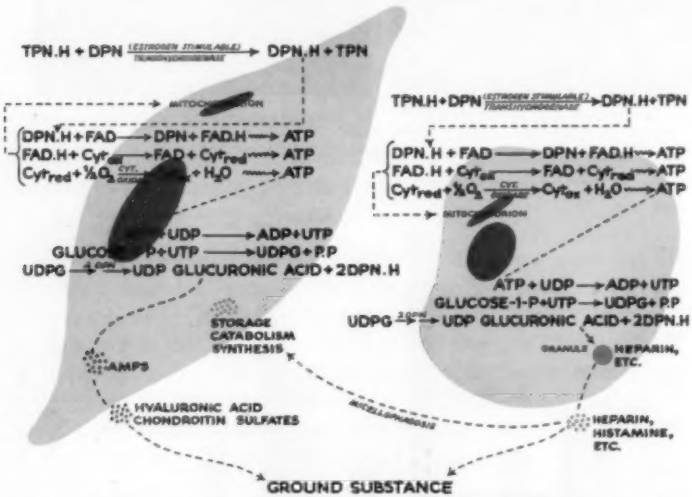


Fig. 4. Effect of Estrogen on Enzyme Systems in Mucopolysaccharide Formation. (Fibroblast and Mast Cell Models.) TPN.H—Reduced Triphosphopyridine Nucleotide; TPN—Triphosphopyridine Nucleotide (Co-enzyme II); DPN.H—Reduced Diphosphopyridine Nucleotide; DPN—Diphosphopyridine Nucleotide (Co-enzyme I); FAD.H—Reduced Flavin Adenine Dinucleotide; FAD—Oxidized Flavin Adenine Dinucleotide; CYT_{ox}—Oxidized Cytochrome; CYT_{red}—Reduced Cytochrome; ATP—Adenosine Triphosphate; ADP—Adenosine Diphosphate; UDP—Uridine Diphosphate; UTP—Uridine Triphosphate; UDPG—Uridine Diphosphate Glucose; UDPGlucuronic—Uridine Diphosphate Glucuronic Acid; Glucose-1-P—Glucose-1-Phosphate; P.P.—Pyrophosphate; AMPS—Acid Mucopolysaccharides.

release the granules into the ground substance.[8,22] The increase in the number of mast cells following estrogens is less in males than in females.[23]

Intravenous Premarin causes an increase in the storage of granules in the mast cells as well as the total number of cells. These, together with a relative decrease of the total count in the number of degranulating mast cells when compared with surgery alone, favor a condition in which the ground substance is better able to withstand the insult of injury. An increased number of storage granules means more trephones are available for the metabolic effects of estrogen. This in turn can alter the mucopolysaccharide content of the ground substance.

What might be the significance in the decreasing ratio of degranulating cells in the Premarin treated cases? Such a finding would mean less histamine and other substances in the tissue and thereby less edema. The result may be visualized as a shift in the sol-gel equilibrium to a more favorable gel state (see Fig. 3). More mast cells can be made to degranulate if tissue water is increased or if hypertonic or hypotonic solutions perfuse the tissue. On the other hand, if hyaluronic acid, which is a basic element of the ground substance, is perfused through the tissue, there is essentially no change in the status of the mast cells.[8] The estrogenic hormone might produce an alteration in the strength of the cell membrane as a result of changes in the membrane—ground substance interface.

In animals given estrogens the following were observed: *a.* an increase in the content of acid mucopolysaccharides, particularly hyaluronic acid in all connective tissues[24,25]; *b.* spreading of fluids in the skin of animals is restricted as is the permeability of small blood vessels[26,27]; *c.* hyaluronic acid production is stimulated in oophorectomized monkeys.[28,29] These findings have been confirmed in our studies of human tissues.

A graphic correlation of the activity of the mast cell and the simultaneous activity of the ground substance is seen in

Figure 3. The effect of a shift in the water content and of mucopolysaccharide polymer has been observed by Slack,[30] and Gersh,[31] as well as by these authors.[2] The metabolites brought by the arterial blood supply, as well as the products that leave by the venous capillaries and lymphatics, help maintain the equilibrium of the ground substance. There are many factors which shift the equilibrium from the water rich state (B) to the water poor state (A) ; for example, in infancy the water content of the tissue is high, and in older ages the content is low.

All these changes influence the mucopolysaccharide water ratio of the ground substance and the larger the polymer or macromolecule, the greater the water binding power. To translate this concept into clinical situations, conjugated estrogens appear to exert a specific beneficial effect, both in regard to the cells and the mucopolysaccharides of the ground substance.

MOLECULAR MECHANISM OF ACTION.

A proposed mechanism whereby Premarin produces these biological and histochemical alterations is helpful and significant. Electron microscopy studies[32,33] have shown that mast cells have both round and rod shaped mitochondria which possess internal cristae. There is a system of membranes near the nucleus which is similar to the lamellar component of the Golgi apparatus, but very little endoplasmic reticulum. Rogers[32] states that their structural features are consistent with appearances to be expected in a cell actively engaged in secretion. Radiosulfate S^{35} isotope studies[34] showed that estrogens stimulate its incorporation into the genital mucosa as well as the activity of the mast cell.

With regard to the inter-relationship between enzymes and hormones, Villee[35] pointed out that estradiol increased by 50 per cent the oxygen consumption of a tissue homogenate in the presence of citric acid as substrate. This also produced a similar increased conversion from isocitrate to alpha-ketoglutarate as noted in the Krebs cycle.

The enzyme systems presumably involved in the intracellular metabolism can be seen in Figure 4. In the case of the isocitric acid, the transhydrogenase reaction was stimulated by estradiol.

$$\text{TPN.H} + \text{DPN} \xrightarrow{\text{transhydrogenase}} \text{DPN.H} + \text{TPN}$$

Interesting enough, thyroxin inhibits this reaction. From the above process, DPN.H becomes available to the cytochrome system of the mitochondrion. This intracellular structure is a chemical factory[36] especially geared for electron transport and oxidative phosphorylation with the production of three molecules of ATP for every one-half molecule of oxygen consumed. Some of the major intermediary steps in this system can be seen in Figure 4.

With the high energy bond phosphate, ATP, now available, another system directly related to mucopolysaccharide production may now be influenced.

$$\text{ATP} + \text{UDP} \longrightarrow \text{ADP} + \text{UTP}$$

The UTP may condense with glucose and proceed to the formation of UDP-glucuronic acid and similarly to form UDP-N-Acetylglucosamine as shown by Roseman.[37] Using cell free extracts of Rous Sarcoma these two substances, UDP-Glucuronic acid and UDP-N-Acetylglucosamine, can be polymerized into low molecular weight hyaluronic acid.[30]

The above sequence of events probably occurs in the mast cells and in the fibroblasts. Asboe-Hansen[38] believes that the mast cell makes hyaluronic acid. Others[6] believe that only the precursors are made in the mast cell.

One of the chief components of the granules of the mast cell is heparin, a sulfated mucopolysaccharide. Such granules are often found in the ground substance and may be ingested into the fibroblasts by the process of micellophagosis. In his excellent paper, Higginbotham[39] describes this process as a collaborative function of fibroblasts, mast cells and ground substance mucopolysaccharides, which serve in the maintenance of the physiological state in the tissues. In the fibroblast, the ingested granules may be stored or metabolized

catabolically to form the small components, or anabolically to form the larger polymer acid mucopolysaccharides. They leave the fibroblasts and enter the ground substance as hyaluronic acid or chondroitin sulphates A, B or C. The controversy as to whether the mast cells or the fibroblasts manufacture the mucopolysaccharides of the ground substance can thus be resolved without compromise. They are both involved in this system, but the trephones of the mast cell may exert a somewhat regulatory effect. Phylogenetically or developmentally, the fibroblasts are present first and thereby make the first mucopolysaccharides. In addition, the rapidity of the cellular response can be better understood in light of the stimulating effect that estrogens have on the TPN.H system.

One might question the source of TPN.H within the cell. The granules of the mast cell have been shown by Sylven[12] to contain ribonucleic acid, RNA. In the manufacture of ribose for RNA, for example, via the direct oxidative pathway or hexose monophosphate (HMP) shunt, TPN.H is formed.[40] Thus the appropriate hydrogen donor is present in adequate quantity to respond on demand to estrogen stimulation.

The ion exchange function of acid mucopolysaccharides[39] offers an intriguing explanation for their protective function. In animals, Weisskopf and Burn[41] showed that Premarin afforded a protective influence to streptococci toxicity; likewise, against toxic agents,[42] the mucopolysaccharides of the connective tissue exerted their effect. It has been observed that the addition of ACTH, the stress hormone, increases the toxicity of some of these agents such as polymyxin. One might then speculate on the beneficial effect afforded by the estrogen stimulation to the connective tissue as working either by a central inhibiting influence on ACTH or by increasing the amount of mucopolysaccharides in the ground substance and thereby neutralizing the local tissue effect of ACTH. Higginbotham[43] performed several experiments with Premarin and also with the respective constituent sulfated estrogens and noted that they complexed with both ACTH and polymyxin B; thus, one is able to conclude that the protective

effect of estrogen is not by way of the general endocrine system but is probably a local cellular and ground substance response.

In medicine, there are many conditions which might be better explained by understanding the importance of the ground substance and cellular response to varying stimuli. Allergy and tissue injury are among the obvious. In hydrops of the labyrinth, there exists an abnormality of vascular permeability of fluid imbalance and frequently the hormonal disturbance of chronic stress. All these abnormalities are often found in disturbances of the ground substance. What role do intracellular enzyme disturbances play in the toxic or traumatic necrobiosis or death of hair cells? The application of such basic studies has the potential to elucidate some of the most perplexing problems in the field of otolaryngology.

SUMMARY.

Histochemical studies on the oral mucosa of human volunteers with surgery plus Premarin and surgery alone present three interesting findings:

1. An increase in the amount of acid mucopolysaccharides, which correlates with the change of the mast cell population.

2. Conjugated estrogens (Premarin Intravenous) appear to exert a protective effect on the mast cell with a resultant increase in total count as well as an increase in granule storage.

3. There is a greater proportional rate of degranulation of the mast cells with surgery alone.

The significance of these changes would seem to indicate that a beneficial effect is exerted on the ground substances under the influence of the conjugated estrogens.

A proposed mechanism whereby the estrogens produce these biological and histochemical alterations is presented in schematic form. This involves a consideration of the molecular and enzyme level of cellular metabolism.

The authors thank Miss Anne Kieffer and Miss Marcia Rew for their able assistance in preparing the sections used in this study. They also thank Miss Laurel Gilliland for the many hours spent in preparing the illustrations.

BIBLIOGRAPHY.

1. Schiff, M., and Brodine, C.: Unpublished data.

2. Schiff, M., and Burn, H. F.: The Effect of Intravenous Estrogens on Ground Substance. *Arch. Otolaryngol.*, 73:43, 1961.

3. Weisskopf, A., and Burn, H. F.: The Ground Substance of the Nasal Turbinates. *Ann. Otol., Rhinol. and Laryngol.*, 67:292, 1958.

4. Downey, H.: The Development of the Histogenous Mast Cells of Adult Guinea Pig and Cat, and the Structure of the Histogenous Mast Cells of Man. *Folia Haematologica*, 16:49, 1913.

5. Riley, J. F.: Pharmacology and Functions of the Mast Cells. *Pharm. Rev.*, 7:267, 1955.

6. Riley, J. F.: The Mast Cells. E&S Livingstone, Ltd., Edinburgh, 1959.

7. Benditt, E. P., Wong, R. L., Arase, M., and Roeper, E.: 5-Hydroxytryptamine in Mast Cells. *Proc. Soc. Exp. Biol. Med.*, 90:303, 1955.

8. Wegelius, O., and Asboe-Hansen, G.: Mast Cells and Tissue Water. Studies on Living Connective Tissue in the Hamster Cheek Pouch. *Exp. Cell. Res.*, 11:437, 1956.

9. Montagna, W.: Histology and Cytochemistry of Human Skin. XI. The Distribution of Beta-Glucuronidase. *Jour. Biophys. Biochem. Cyt.*, 3:343, 1957.

10. Benditt, E. P., and Arase, M.: An Enzyme in Mast Cells with Properties Like Chymotrypsin. *Jour. Exp. Med.*, 110:451, 1959.

11. Montagna, W., and Noback, C. R.: Localization of Lipids and Other Chemical Substances in the Mast Cells of Man and Laboratory Mammals. *Anat. Rec.*, 100:535, 1948.

12. Sylven, B.: On the Topographical Cytochemistry of Tissue Mast Cells. Connective Tissue Symposium. Blackwell Scientific Publications, Oxford, p. 27, 1957.

13. Asboe-Hansen, G.: Endocrine Control of Connective Tissue. *Amer. Jour. Med.*, 26:113, 1959.

14. Asboe-Hansen, G.: Autoradiographic Evidence of Cortisone Action on Mast Cells in Experimental Skin Tumors. *Cancer Res.*, 14:94, 1954.

15. Asboe-Hansen, G.: Effect of the Adrenocorticotropic Hormone of the Pituitary on Mesenchymal Tissues. *Scand. Jour. Clin. Lab. Invest.*, 2:271, 1950.

16. Zachariae, L., and Moltke, E.: Influence of Hydrocortisone (Compound F) on Mast Cells of Normal Skin and Healing Wounds in the Rabbit. *Acta Endocr.*, 16:300, 1954.

17. Paff, G. H., and Stewart, R.: Free Wandering Cells and Cortisone. *Proc. Soc. Exp. Biol. Med.*, 83:591, 1953.

18. Goth, A., Allman, R., Merritt, B., and Holman, J.: Effect of Cortisone on Histamine Liberation Induced by Tween in the Dog. *Proc. Soc. Exp. Biol. Med.*, 78:848, 1951.

19. Asboe-Hansen, G., and Inversen, K.: Influence of Thyrotrophic Hormone on Connective Tissue. Pathogenetic Significance of Mucopolysaccharides in Experimental Exophthalmos. *Acta Endocr.*, 8:90, 1951.

20. Wegelius, O., and Asboe-Hansen, G.: Hormonal Effects on Mast Cells. Studies on Living Connective Tissue in the Hamster Cheek Pouch. *Acta Endocr.*, 22:157, 1956.

21. LARSEN, G.: The Mast Cells in the Uveal Tract of the Eye and Changes Induced by Hormones and Avitaminosis-C. *Amer. Jour. Ophthal.* (In Press.)

22. ASBOE-HANSEN, G., and WEGELIUS, O.: Histamine and Mast Cells; Studies on Living Connective Tissue in the Hamster Cheek Pouch. *Acta Physiol. Scand.*, 37:350, 1956.

23. ARVEY, L.: Effect of Estrogen on the Mast Cells of the White Mouse. *Nature* (Lond.), 175:1, 506, 1955.

24. CHAIN, E., and DUTHIE, E. S.: Identity of Hyaluronidase and Spreading Factor. *Brit. Jour. Exp. Path.*, 21:324, 1940.

25. OGSTON, A. G., PHILPOT, J. ST. L., and ZUCKERMAN, S.: Observations Related to the Swelling of Sexual Skin in Rhesus Monkeys. *Jour. Endocr.*, 1:231, 1939.

26. COOPER, D. J., and SCHMIDT, A.: The Effect of Sex Hormones on the Spreading Reaction and the Reconstitution of the Connective-Tissue Barrier in the Mouse. *Acta Pharmacol. et Toxicol.*, 13:155, 1957.

27. TAYLOR, H. M., and SPRUNT, D. H.: Increased Resistance to Viral Infection as a Result of Increased Fluid in Tissues. *Jour. Exp. Med.*, 78:91, 1943.

28. ALLEN, E.: Menstrual Cycle of Monkey, Macacus Rhesus: Observations on Normal Animals, Effects of Ovarian and Placental Extracts Into Spayed Animals. *Contrib. Embryol.*, 19:1, 1927.

29. ZUCKERMAN, S., VON WAGENEN, G., and GARDINER, R. H.: The Sexual Skin of the Rhesus Monkey. *Proc. Zool. Soc.*, London, S.A., 108:385, 1938.

30. SLACK, H. G. B.: Some Notes on the Composition and Metabolism of Connective Tissue. *Amer. Jour. Med.*, 26:113, 1959.

31. GERSH, I., and CATCHPOLE, H. R.: Nature of Ground Substance of Connective Tissue. *Perspectives Biol. Med.*, 3:282, 1959-60.

32. ROGERS, G. E.: Electron Microscopy of Mast Cells in the Skin of Young Mice. *Exp. Cell. Res.*, 11:393, 1956.

33. STOECKENIUS, W.: Zur Feinstruktur der Granula Menschlicher Gewebsmastzellen. *Exp. Cell. Res.*, 11:656, 1956.

34. ZACHARIAE, F.: Autoradiographic (S^{35}) and Histochemical Studies of Sulfomucopolysaccharides in the Rabbit Uterus, Oviducts and Vagina; Variations Under Hormonal Influence. *Acta Endocr.*, 29:118, 1958.

35. VILLEE, C. A.: Enzymes in Health and Disease. Edited by D. M. Greenberg and H. A. Harper. Springfield, Ill. Charles C. Thomas, 1960, Chapter 5.

36. GREEN, D. E., and HATEFI, Y.: The Mitrochondrion and Biochemical Machine. *Science*, 113:3445, 1961.

37. ROSEMAN, S.: Mucopolysaccharides Metabolism. *Amer. Jour. Med.*, 26:749, 1959.

38. ASBOE-HANSEN, G., and ZACHARIAE, L.: Mast Cell Changes Induced by Hydrocortisone Acetate in Experimental Skin Papillomas in Mice. *Acta Path. Microbiol., Scand.*, 37:145, 1955.

39. HIGGINBOTHAM, R. D.: On the Participation of Micellophagosis in the Resistance of Tissue to Injurious Agents. *Int. Arch. Allergy*, 15:195, 1959.

40. HARPER, H. A.: Review of Physiologic Chemistry, pp. 188, 7th Edition. Lange Medical Publications, Los Altos, Calif., 1959.

41. Weisskopf, A., Burn, H. F., and Schoenholz, W. K.: Protective Action of Estrogen Against Streptococcal Infection in Mice and Its Relation to Connective Tissue. The Laryngoscope, 71:788-799, July, 1961.

42. Higginbotham, R. D.: Mast Cells, Heparin and Toxic Agents. *Fed. Proc.*, 17:516, 1958.

43. Higginbotham, R. D.: Personal Communication.

SIXTH INTERNATIONAL CONGRESS OF AUDIOLOGY.

The Sixth International Congress of Audiology will be held in Leyden, The Netherlands, September 5-8, 1962.

President: Prof. Dr. H. A. E. van Dishoeck;

Secretary: Dr. A. Spoor.

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Official languages of the Congress are: English, French, German and Spanish. Working languages will be: English and French.

For further information address the secretariat, Ear-Nose-Throat Department, Academisch Ziekenhuis, Leyden (The Netherlands).

THE PUBLISHED WORKS OF DOCTOR ROBERT C. MARTIN.*†

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This paper would have been more properly entitled, "The Forward Look" because it concerns the writings of a man who always kept his eye on the future while adding to the knowledge of the present. Dr. Robert C. Martin contributed 19 papers to the medical literature between the years 1929 and 1955, and each one has added some important concepts to our specialty. There are some general statements which can be made about all of Dr. Martin's writings. Most noteworthy are his style and manner of presentation. Each paper is well organized, straight forward and understandable. It never leaves the reader in doubt about its basic purpose, and the conclusions are logical and of practical value. Second, we find impeccable honesty in reporting his results—failures along with the successes. No claims of personal achievements are made anywhere, but full acknowledgments are found throughout, giving credit to others for their collaboration.

Now let us look at the work itself. It might be divided into three main categories:

1. Papers concerning the injury and repair of the facial nerve.

2. Papers concerning fenestration of the horizontal semicircular canal, and

3. Miscellaneous topics.

Injury and Repair of the Facial Nerve.

At the turn of the Century, Faure and Korte made their

*Read at the Scientific Program of Otorhinolaryngology in Honor of Robert C. Martin, M.D., University of California School of Medicine, San Francisco, Calif., April 15, 1961.

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first attempts at repairing the injured facial nerve by anastomosing its distal segment to the proximal trunks of the spino-accessory and hypoglossal nerves, respectively. This was done below the stylomastoid foramen. Results were poor, since in the case of spino-facial anastomosis half of the face twitched with shoulder movements, and in the case of hypoglosso-facial suture it twitched grotesquely during speech. In 1922, Ney presented an extensive and detailed review of facial nerve anatomy, in which he worked out a step-by-step procedure on cadavers, for facial nerve decompression. Sterling Bunnell reported a case in 1927 in which he straightened the intratympanic course of the nerve by lifting it out of its bed. and sutured the injured ends together.

In May of 1929, Dr. Martin was asked to consult on a case of complete facial paralysis following simple mastoidectomy. Inspired by the work of Bunnell, he took the patient to surgery and became the first otolaryngologist to perform an end-to-end anastomosis of the facial nerve in its intra-temporal course. The patient regained facial symmetry, food no longer trickled from the corner of her mouth, and she was able to close her left eye for the first time since her mastoidectomy 18 months before. Dr. Martin then wrote his well-known paper called, "Intra-temporal Suture of the Facial Nerve," which he read before the Pacific Coast Oto-Ophthalmological Society in September, 1930, and which was published in the Archives in February, 1931. In it, he described in detail the anatomy of the facial canal, the nature of surgical injury, the optional time for repair, and the exact operative technique. He exposed the nerve over its entire intra-temporal course, located the point of injury, excised the neuroma which had formed, lifted the nerve out of its bed between the oval window and horizontal canal as well as in the vertical portion, and was able to suture the ends together. This work, pioneered by Dr. Martin exactly 32 years ago, stands unchallenged and unaltered as the cornerstone of facial nerve surgery, as useful today as it was three decades ago.

In the following years much was done to refine and categorize facial nerve work. In 1936, Dr. Martin published a paper describing the three useful methods of repair, giving

case reports for each type; namely, decompression and neurolysis, end-to-end anastomosis, and interposition of a homologous nerve graft by the method of Ballance and Duel. He also outlined his refinements in operative technique, giving a step-by-step description of how the facial canal is exposed and laid open with the least amount of trauma to the nerve.

By 1940 16 patients had been restored to facial symmetry and adequate facial function. Four noteworthy cases were reported in the A.M.A. Archives. One was an 18-month-old boy in whom a 1.5 cm. long piece of sural nerve graft had to be interposed, and in whom almost complete regeneration had occurred in a period of only two months. Two other cases also illustrated the remarkable power of nerve regeneration in young children. One six-year-old girl had almost complete return of function of her frontalis muscle which occurs only very rarely. The other was a two-year-old boy in whom return was so complete after insertion of a sural nerve graft that three months later complete facial symmetry was restored.

When the Naftziger Number of the Journal of Nervous and Mental Disease appeared in May, 1944, to honor the late Dr. Howard Naftziger, it included an article by Dr. Robert C. Martin on the repair of peripheral injuries of the facial nerve. Rather than dwell on his own achievements in this field, he saw fit to discuss the shortcomings of any type of VIIth nerve repair. The chief ones, of course, are associated movements, tics, and lack of response to emotional expression.

Keeping abreast with new techniques and lifelong learning are cardinal signs of a conscientious physician. Soon after electromyography became established as a diagnostic tool, Dr. Martin could see its application in determining the operability of facial nerve palsies. In May, 1951, he read a paper entitled, "Prognosis in Traumatic Peripheral Facial Paralysis," before the Pacific Coast Oto-Ophthalmological Society. In it, he proposed that electromyography, though secondary in importance to an accurate history and examination, is an important adjunct to determining the prognosis of nerve regeneration. He states that fibrillation potentials, or reaction of degeneration, does not occur until 12-14 days after peripheral injury.

If these fibrillation potentials are few in number and quite scattered, the anatomical interruption may not be complete. Four cases are reported: three of surgical trauma and one of facial paralysis of sudden onset 17 years after radical mastoidectomy. In all cases, electromyography showed development of only minimal fibrillation potentials. Surgery was withheld, and all cases showed excellent spontaneous recovery over a period of several months. It was concluded that more nerves will be saved, to resume their natural function, through careful evaluation of the history, physical findings and electromyographic results, than by hasty surgical intervention.

In his most recent paper, Dr. Martin discussed the "Late Results of Facial Nerve Repair." The three types of intratemporal facial nerve repair—decompression neurolysis, end-to-end anastomosis, and nerve grafting—are compared on the basis of long-term results. Six of the case follow-ups ranged an incredible 18-26 years after surgical repair. This includes the notable case of 1929. It was concluded that nerve grafts, though showing rapid initial return of tonus and motor function, give little improvement after one year postoperatively. On the other hand, end-to-end anastomoses and neurolyses show improvement over the years, although complete normality is never attained.

It may be clearly seen from the above, that Dr. Martin deserves to be called the father of facial nerve surgery.

Fenestration of the Horizontal Canal.

Although only two papers have been published by Dr. Martin on this subject, his work in this field has been notable. He was the first man in Northern California to perform the operation, and this only after 18 months of arduous labor in temporal bone dissections. While never fearful to utilize new thoughts and new techniques, he always showed wisdom and conservatism. When Lempert's work gained popular fame in the early 1940's, an article appeared in the Reader's Digest acclaiming fenestration as a panacea for otosclerosis. Dr. Martin promptly countered with a paper read before the California Medical Association in 1945 giving the historical background, critical indications for surgery, and expected

results. He cautioned against overenthusiasm and advised a very careful selection of patients. In doing this, he did not detract from the importance of the operation, but lauded it as the first and most significant advance in otology during his lifetime. By 1948, he was able to report an impressive series of 122 fenestrations performed. He analyzed his results on the basis of his failures, not his successes, and classified them into bony closures, fibrous closures, and failure to improve initially. He emphasized Day's admonition to clean the skin flap meticulously of all bony fragments and recommended the "dome" technique for removal of endosteum together with bone to create the fenestra. Vertigo, tinnitus, and wound healing are also discussed in this paper. Dr. Martin continued his highly skilled work in fenestration until the advent of stapes surgery.

Miscellaneous Topics.

In the ten remaining papers by Dr. Martin a variety of subjects are discussed. Several of these cannot be passed bv without mention.

Dr. Martin and Dr. F. C. Cordes of the University of California Department of Ophthalmology were the first men on the West Coast to perform the Toti-Mosher operation for chronic suppurative dacryocystitis. Before 1929, most cases were treated by dacryocystectomy while a small percentage of patients were relieved by simple probing. In their article, Martin and Cordes gave a detailed description of the Toti-Mosher operation and reported 14 cases so treated.

In 1942, a most unusual report appeared in the Journal of the American Medical Association under Dr. Martin's name: "Atlas-Axis Dislocation Following Cervical Infections." In it he discussed the pathological changes which can take place in cases of severe infections of the neck. These are hyperemia of the bone due to infection, vertebral decalcification, loosening of the ligamentous attachments, and slipping forward of the lateral articular facets of the axis. The result is a dislocation and locking of the atlas-axis joint. The treatment consists of reduction of the dislocation, followed by cervical traction.

Four such cases were presented, all of which preceded the antibiotic era by several years.

Other papers by Dr. Martin illustrate his versatility, as they encompass such topics as "Osteomyelitis of the Skull in Sinusitis," "The Treatment of Suppurative Otitis Media," and "Atypical Facial Neuralgia."

Perhaps his most significant work was not a scientific paper, but his Presidential Address at the Fifty-fourth meeting of the Triological Society in 1950. It was addressed to the young man about to enter the field of medicine. It gave a strong and effective answer to the undercurrent of radicalism and socialistic reform which was already gaining momentum these 12 years ago. In closing, permit me to quote a few lines from this address, which will illustrate that Dr. Martin's writings and principles are as true and applicable today as they were when first set on paper. I quote:

"I do not believe that a bureaucratic system with the Utopia of financial security and regular working hours appeals to the prospective physician. It robs medicine of its greatest burden—and its charm—which is individual responsibility. I for one do not want the next shift to take over a patient about whom I am worried!

"The pendulum may swing to lower standards and loss of individuality, but the people cannot be fooled all of the time, and some day it will swing back and finally settle to a midpoint of common sense. It may be after your time before another Magna Carta can be forced from the hands of reluctant, privilege-seeking politicians. As surely as I know human nature, after years of practice, you will find that both the donor of service, the doctor, and the recipient of service, the patient, will eventually fight to retain or regain their individuality and self-respect."

What could be more apropos today?

BIBLIOGRAPHY.

1. MARTIN, R. C., and CORDES, F. C.: Toti-Mosher Operation in Obstruction of the Nasolacrimal Duct. *Calif. and Western Med.*, 31, Oct., 1929.

2. MARTIN, R. C.: Intratemporal Suture of the Facial Nerve. *Arch. Otolaryngol.*, 13:259-264, Feb., 1931.

3. MARTIN, R. C.: Streptothrix Infections of the Mastoid Middle, and External Ear. *Ann. Otol., Rhinol. and Laryngol.*, 40:1171-1179, Dec., 1931.

4. MARTIN, R. C.: Surgical Repair of the Facial Nerve. *Arch. Otolaryngol.*, 23:458-466, April, 1936.

5. MARTIN, R. C.: Malignancies of the Nose and Throat. *Ann. Otol., Rhinol. and Laryngol.*, 47:512-522, June, 1938.

6. MARTIN, R. C.: Recent Experiences with Operations on the Facial Nerve. *Arch. Otolaryngol.*, 32:1071-1075, Dec., 1940.

7. MARTIN, R. C.: Atlas-Axis Dislocation Following Cervical Infection. *Jour. A.M.A.*, 118:874-875, March, 1942.

8. MARTIN, R. C.: Atypical Facial Neuralgia. *Arch. Otolaryngol.*, 35: 735-739, May, 1942.

9. MARTIN, R. C., DANNO, D., and SKELLY, J. F.: (Progress in Otolaryngology) Suppurative Otitis Media and Its Complications. *Arch. Otolaryngol.*, 37:727-736, May, 1943.

10. MARTIN, R. C.: Osteomyelitis of the Skull in Sinusitis. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc., Inc.*, 1943.

11. MARTIN, R. C.: Repair of Peripheral Injuries of the Facial Nerve. *Jour. Nerv. and Mental Dis.*, 99 (Naffziger Number), May, 1944.

12. MARTIN, R. C.: Fenestration Operation for Deafness. *Calif. and Western Med.*, 62, June, 1945.

13. MARTIN, R. C.: Fenestration of the Labyrinth. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc., Inc.*, 1948.

14. MARTIN, R. C.: Medical Education (Presidential Address). THE LARYNGOSCOPE, 60:601-605, July, 1950.

15. MARTIN, R. C.: Prognosis in Traumatic Peripheral Facial Paralysis. THE LARYNGOSCOPE, 61:1004-1009, Oct., 1951.

16. MARTIN, R. C.: Bell's Palsy. *Arch. Otolaryngol.*, 55:405-409, April, 1952.

17. MARTIN, R. C.: Late Results of Facial Nerve Repair. *Ann. Otol., Rhinol. and Laryngol.*, 64:859-870, Sept., 1955.

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PROTECTIVE ACTION OF ESTROGEN AGAINST STREPTOCOCCAL INFECTION IN MICE, AND ITS RELATION TO CONNECTIVE TISSUE.*†‡

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All otolaryngologists are beset by problems of recurrent infections which do not respond to ordinary therapy. The problem of response to infection, although considerably mitigated by the advent of the antibiotics, is still not solved. We still depend primarily upon mobilization of local and systemic antibacterial mechanisms. Continued experience with antibiotics has emphasized the need for understanding these mechanisms and finding means for their improvement. Increasing drug reactions and formation of unresponsive mutants have emphasized the need for re-evaluation of the original optimism in the "brave new sterile world" of antibiotics.

Recent work has related developments in connective tissue physiology to the problems of otolaryngology.[1-4] These studies have suggested another approach in the understanding of disease and opened new avenues. The present work on streptococcal infection in mice arose from this background.

Previous experiments with rabbits and mice have shown that estrogens have a protective action against infection by both viruses and bacteria.[5-10] The mechanism of this protection is not clearly understood; however, when viewed in the light of connective tissue physiology, certain broad principles

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develop which may lead to a rational explanation of these responses.

It has been shown repeatedly that estrogens cause an increased output of the acid mucopolysaccharides of the ground substance.[11-16] This is associated with decreased subcutaneous spread of solutions, particulate material and bacteria.[6,8-11] It is a physical indication of the increased turgidity of the ground substance due to increase in the highly polymerized mucopolysaccharides. A decrease in capillary permeability has likewise been shown.[10,16] The participation of other protective factors is suggested by recent work which demonstrated an increased leukocytic response to infection of rabbit uteri following the use of estradiol.[17]

The present work constitutes a re-examination of the protective action of estrogen and an attempt at clarification of its basic mechanisms in the light of newer histochemical and biochemical knowledge.

EXPERIMENTAL DATA.

A total of 1100 Swiss-Webster mice, from a single source, and weighing from 18 to 20 grams, was used. The animals were kept under identical experimental conditions and fed a standard diet of pellets and water *ad libitum*. Groups of mice never exceeded ten animals per cage. Premarin (R), a preparation of conjugated estrogens derived from pregnant mare's urine, was used as the estrogenic hormone.

A streptococcus, received from Doctor Rebecca Lancefield at the Rockefeller Institute, was employed throughout these studies. It was a lyophilized beta-hemolytic streptococcus, Group A, Type 9, which was originally isolated from a human rheumatic fever case. This strain, designated as RP 124, was reconstituted in nutrient broth (Difco) and an 18-hour culture was standardized to contain 5×10^5 colony-forming units. Its mouse virulence was established by LD_{50} determination[18] at appropriate time intervals. Whenever passage through media resulted in loss of virulence, a single mouse passage of the organism was carried out to re-establish the original LD_{50}.

The streptococcal dose for individual experiments was adjusted so that control animals would succumb in approximately 24 hours.

Preliminary experiments showed that it was difficult to produce a consistently high level of infection when streptococci were given intranasally and the intraperitoneal route

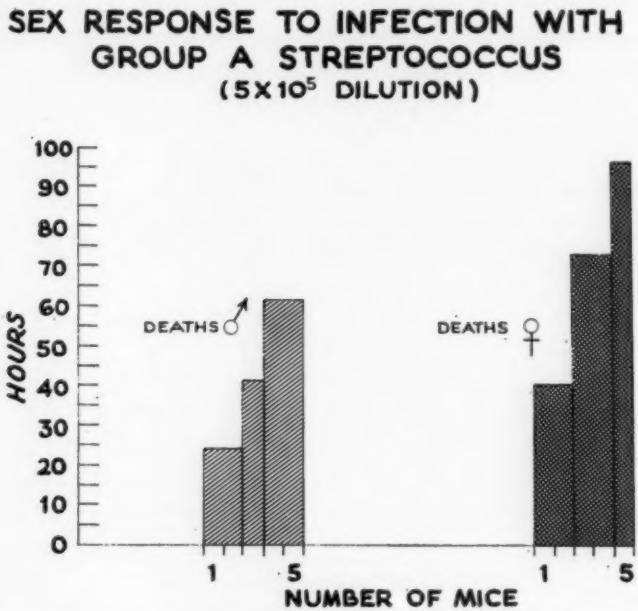

Fig. 1. Death rates of male and female mice (Swiss-Webster) inoculated with 5 x 10^5 units of Group A streptococcus.

was chosen. In the early experiments, Premarin (R) was dissolved in normal saline. It was given intraperitoneally one week before challenge with streptococci, or intravenously on the day of challenge. There was little protection with either route. Previous work had shown the effectiveness of estradiol in sesame oil.[7] An oil adjuvant (Freund's Incom-

plete)[19] was tested as a vehicle for the estrogen and was found to give an optimum protective effect. This estrogen-adjuvant mixture was used in all subsequent experiments.

Figure 1 is a graphic representation of one of the earlier titration experiments to determine the effect of 5 x 10^5 organisms on normal mice. The average time to death for males was 41.6 hours as compared with 64 hours for females. A

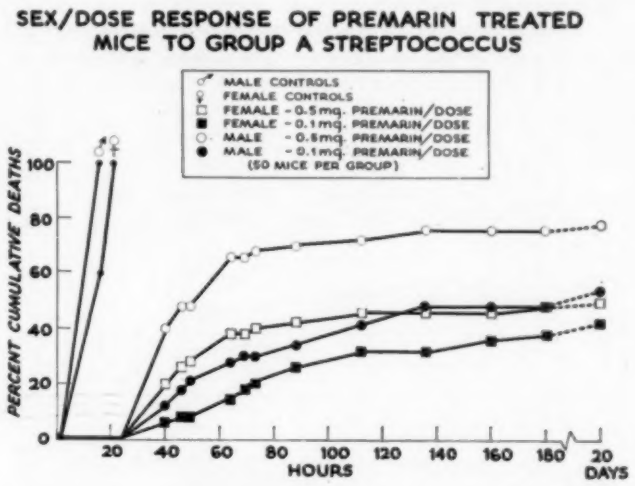

Fig. 2. Cumulative deaths of mice (Swiss-Webster) receiving two weekly doses of Premarin in adjuvant and challenged one week later with 5 x 10^5 units of Group A streptococcus.

similar difference in sex response was constant in all of the experiments.

The percentage of cumulative deaths of mice which had received two weekly doses of estrogen and were challenged one week later with 5 x 10^5 units of streptococci is shown in Figure 2 and in Table I. In the groups receiving streptococci only, all male mice were dead at 16 hours, and all female mice were dead at 21 hours. At 24 hours, there were no deaths in any of the mice which had been protected with

TABLE I.

Survival Rate of Mice (Swiss-Webster) Receiving Two Weekly Doses of Premarin (R) in Adjuvant and Challenged One Week Later with 5×10^5 Units of Group A Streptococcus (50 Mice Per Group).

Sex and Dose	Per Cent Survivors at Hours														
	16	21	24	40	46	49	64	69	73	88	112	136	160	180	480 (20 Day)
Male Controls	0														
Female Controls	40	0													
Female 0.5 mg. Premarin/Dose	100	100	100	80	74	72	62	62	60	58	54	54	54	52	50
Male 0.5 mg. Premarin/Dose	100	100	100	60	52	52	34	34	32	30	28	24	24	24	22 (1*)
Female 0.1 mg. Premarin/Dose	100	100	100	94	92	92	86	82	80	74	68	68	64	60	58 (1*)
Male 0.1 mg. Premarin/Dose	100	100	100	88	82	78	72	70	70	66	58	52	52	52	46 (1*)

*Secondary Infection.

estrogen. The levelling of the curve for protected mice indicated that the infection had become less lethal about the third day. There were seven deaths between 180 hours (7½ days) and 20 days and three of these were from secondary infections. This chart shows that the dosage of estrogen was also important. In preliminary work, doses of 1 mg. or of 0.05 mg. of estrogen had shown little protective effect. In this experiment, it was found that 0.1 mg. of estrogen had a

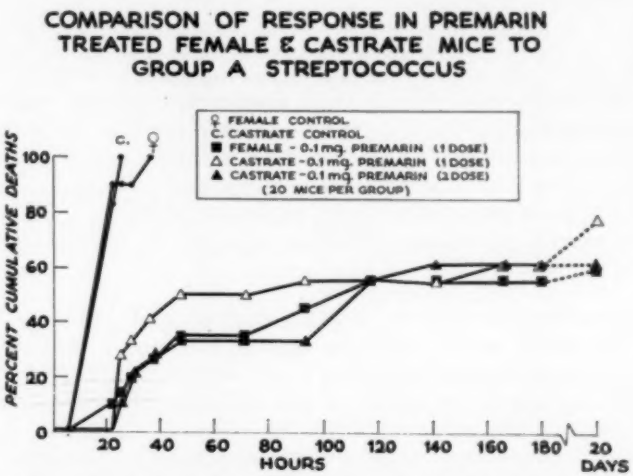

Fig. 3. Cumulative deaths of female and castrate mice (Swiss-Webster) receiving Premarin in adjuvant and challenged one week later with 5 x 10^4 units of Group A streptococcus.

greater protective effect than 0.5 mg. in both sexes. Variation above and below this critical level increased the mortality. It is also evident from this chart that the degree of protection varied with the sex. The females survived longer than the males in both of the dosages given.

Since male mice showed less protection than females, a group of males were castrated one month before estrogen treatment to evaluate the role of the testicular hormones. One dose of estrogen was given to the female and to one of the

castrate groups and two weekly doses to the other castrate group (see Fig. 3). Of the unprotected control mice, all of the castrates and 90 per cent of the females were dead at 25 hours. In the protected groups, there was little difference between the death rates of the females and the castrate males for the first seven days. The death rate in the castrate group receiving only one dose of estrogen, rose during the next 13 days.

Although the general trend of these results is evident and has been reproducible consistently, it should be emphasized that the time/mortality relationship varies somewhat from one experiment to the next. The slight fluctuation in virulence of the streptococci during their growth in artificial media, was probably the main factor responsible for this variation. Appropriate dilution of the infecting agent compensated for this fluctuation, so that the control animals in each experiment showed very similar death responses in all cases. In the light of this adjustment, different experiments were entirely comparable.

The occurrence of three deaths, due to secondary infections, as mentioned above, was investigated further. It was felt that these mice might have been more susceptible to secondary infections due to the stress of the original infection or due to "rebound phenomenon" after the excess estrogen was depleted. The survivors of each of the four groups were pooled at the end of 20 days (see Fig. 2) and separated into two groups of six mice each. The first group received 0.1 mg. of estrogen weekly for four weeks, and the second group remained untreated. Only one mouse in the four groups of mice receiving additional estrogen died, as against six mice in the four groups of untreated mice. All of these deaths were due to Gram positive rods, diphtheroid or pneumococcal organisms as determined by impression smears of livers and spleens. No streptococci were found.

Since it has been shown that one of the actions of estrogen is on the ground substance in the connective tissue, there was an attempt to correlate the connective tissue response with the death rates of the mice. The most easily obtained

TABLE II.

Survival Rate of Female and Castrate Mice Receiving Premarin (R) in Adjuvant and Challenged One Week Later with 5 x 10^4 Units of Group A Streptococcus (20 Mice Per Group).

Sex and Dose	Per Cent Survivors at Hours												
	6	22	25	29	38	47	71	93	117	141	166	180	480 (20 Day)
Female Controls	0	10	10	10	0								
Castrate Controls	0	17	0										
Female 0.1 mg. Premarin/1 Dose	0	90	85	80	73	65	65	55	45	45	45	45	40
Castrate 0.1 mg. Premarin/1 Dose	0	0	72	67	59	50	50	45	45	45	39	39	23
Castrate 0.1 mg. Premarin/2 Dose	0	0	89	78	73	67	67	67	45	39	39	39	39

source of biopsy material was the ear. This was removed, using a light ether anesthesia. It was found that the amount of acid mucopolysaccharides was scanty and variable and did not show clear results.

It has been demonstrated that intracardiac injection of saline Premarin in hamsters caused an increase in connective tissue ground substance in the cheek pouch.[16] This increase was noted within 30 minutes, reached its peak in six to eight hours and subsided slowly, returning to normal in less than 24 hours. A male and female hamster, each weighing about 100 grams, were given 0.5 mg. of Premarin in adjuvant, intraperitoneally, and tissue samples of the cheek pouch were taken at intervals. It was found that there was no connective tissue response at the end of six hours. There was a moderate increase in the acid mucopolysaccharides at three days, and the increase continued up to the sixth day. The large amount of acid mucopolysaccharides in the connective tissue remained constant, and on the eighth day, another dose of estrogen was given. The amount of acid mucopolysaccharides remained constant until the fourteenth day when the experiment was discontinued. Another hamster was given the adjuvant alone and showed a small increase of mucopolysaccharides which was probably due to the stress of a local infection and the repeated biopsies.*

DISCUSSION.

These experiments have presented certain facts and have raised new questions. The often made observation that female mice are less susceptible to the lethal effects of streptococci has been confirmed in these studies. This might be explained by the hormonal factor since there was a closeness of response between the castrate males and the female mice.

Estrogen had a significant protective action in all experiments within certain dosage limits. The reason for this optimal dose is not yet clear but does suggest that the pituitary may be involved. It is known that large doses of estrogen will

*The authors are grateful to Miss Anne Kieffer and Miss Marcia Rew for their enthusiastic assistance in these experiments.

depress the "tropic" hormones of the pituitary. This may disturb the equilibrium involving other endocrine glands and cause a depression of "anti-lethal" factors. This would explain the lessening of protection when 1 mg. and 0.5 mg. of estrogen were administered.

Histochemically, the amount of acid mucopolysaccharides showed a rapid peaking with a slower rate of fall when using estrogen in saline. Since the critical period for streptococcal invasion may be reached at a time when the action of Premarin in saline has subsided, the lack of protection with this method of administration can be explained. When using Premarin in an oil adjuvant, preliminary histochemical studies on the hamster suggest that the increase in acid mucopolysaccharides starts after 24 hours, reaches its peak in four to six days and remains constant for a further period. This is probably due to a slow release of estrogen from the oil depot since traces of oil were found in mice which were autopsied seven weeks after the original injection. Thus, the dosage of 0.05 mg. of estrogen is probably released in too small an amount and too slowly to give a protective effect.

It has been shown that an increase in acid mucopolysaccharides prevents spreading of bacteria and toxins. Estrogen aids in this process by creating longer polymers and forming a more resistant gel in the ground substance. This increase is probably only one of many actions which explain the protection of estrogens against infection. There are other factors which must be investigated before this effect can be completely explained. These include endocrine gland relationships, changes in cellular and acellular humoral constituents, and the effect on the reticuloendothelial and lymphopoietic systems which are assumed to be a site of antibody formation.

The possibility of a "rebound effect" with depletion of estrogen in the tissues is suggested by the increased death rate late in the experiment. This was prevented by continued administration of Premarin. This effect may be due to a refractory phase in the tissues which requires more than normal amounts of estrogen, or may indicate an actual lack of hormones due to suppression of pituitary activity.

It must be emphasized that this work has no therapeutic value at present. It is felt that these experiments have laid the ground work for further study. Is this "protective effect" a true hormonal action or a side reaction not necessarily related to alterations of the secondary sex characteristics? This would be of particular importance in the problem of infection in pre-pubertal children. For otolaryngologists, a solution to this problem would be of value.

SUMMARY.

1. In spite of the widespread use of antibiotics, infection continues to be a problem to otolaryngologists. Basic investigation of the natural antimicrobic factors is necessary since many resistant strains of bacteria are now found.

2. The protective action of Premarin (R), a preparation of conjugated estrogens, against virulent streptococcal infection in mice has been investigated. A significant relationship between the estrogen and the ability to survive lethal infection has been found.

a. In both sexes, Premarin (R) had a marked protective effect within certain dosage limits.

b. The death rate in female mice was consistently less than in male mice.

c. Castration of males resulted in a significant increase in survival, and their death rates approached those of the females.

3. It is postulated that this antimicrobial effect of estrogen is related to changes in the connective tissue. Further work on this phase is planned to clarify this protective action.

BIBLIOGRAPHY.

1. Weisskopf, A., and Burn, H. F.: The Ground Substance of the Nasal Turbinates. *Ann. Otol., Rhinol. and Laryngol.*, 67:292-304, 1958.

2. Weisskopf, A., and Burn, H. F.: Histochemical Studies of the Pathogenesis of Nasal Polyps. *Ann. Otol., Rhinol. and Laryngol.*, 68:509-523, 1959.

3. Schiff, M.: Juvenile Nasopharyngeal Angiofibroma. A Theory of Pathogenesis. The Laryngoscope, 69:981-1016, 1959.

4. Weisskopf, A.: Connective Tissue: A Synthesis of Modern Thought and Its Impact on the Understanding of Nasal Disease. The Laryngoscope, 70:1029-1059, 1960.

5. Sprunt, D. H., and McDearman, S.: Studies on the Relationship of Sex Hormones to Infection. III. A Quantitative Study of the Increased Resistance to Vaccinial Infection Produced by the Estrogenic Hormone and Pseudopregnancy. *Jour. Immun.*, 38:81-95, 1940.

6. Sprunt, D. H., and McDearman, S.: Studies on the Relationship of Sex Hormones to Infection. *Endocrinology*, 27:893-898, 1940.

7. Von Haam, E., and Rosenfeld, I.: The Effect of the Various Sex Hormones Upon Experimental Pneumococcus Infections in Mice. *Jour. Infect. Dis.*, 70:243-247, 1942.

8. Taylor, H. M., and Sprunt, D. H.: Increased Resistance to Viral Infection as a Result of Increased Fluid in Tissues. *Jour. Exp. Med.*, 78:91-97, 1943.

9. Lurie, M. B., Abramson, S., and Allison, M. J.: Constitutional Factors in Resistance to Infection. I. The Effect of Estrogen and Chorionic Gonadotropin on the Course of Tuberculosis in Highly Inbred Rabbits. *Amer. Rev. Tuberc.*, 59:168-185, 1949.

10. Lurie, M. B., Abramson, S., Heppleston, A. G., and Allison, M. J.: Constitutional Factors in Resistance to Infection. III. On the Mode of Action of Estrogen and Gonadotropin on the Progress of Tuberculosis. *Amer. Rev. Tuberc.*, 59:198-218, 1949.

11. Lurie, M. B.: Mechanisms Affecting Spread in Tuberculosis. *Ann. N. Y. Acad. Sc.*, 52:1074-1090, 1950.

12. Bachman, C., Collip, J. B., and Selye, H.: The Effects of Prolonged Oestriol Administration Upon the Sex Skin of Macaca Mulatta. *Proc. R. Soc.*, Ser. B, 117:16-21, 1935.

13. Cooper, D. J., and Schmidt, A.: The Effect of Sex Hormones on the Spreading Reaction and the Reconstitution of the Connective-Tissue Barrier in the Mouse. *Acta Pharm. Tox.*, 13:155-168, 1957.

14. Schmidt, A.: The Influence of Cortisone and Oestradiol on the Amounts of Hexosamine and Water in the Skin. *Acta Pharm. Tox.*, 14:350-358, 1958.

15. Hvidberg, E., and Schou, J.: The Effect of Oestradiol Monobenzoate on Connective Tissue, Especially on Subcutaneous Absorption. *Acta Pharm. Tox.*, 15:207-216, 1959.

16. Schiff, M., and Burn, H. F.: The Effect of Intravenous Estrogens on Ground Substance. *Arch. Otolaryngol.* In Press.

17. Hawk, H. W., Turner, G. D., and Sykes, J. F.: The Effect of Ovarian Hormones on the Uterine Defense Mechanism During the Early Stages of Induced Infection. *Amer. Jour. Vet. Res.*, 21:644-648, 1960.

18. Reed, L. J., and Muench, H.: A Simple Method of Estimating Fifty Per Cent Endpoints. *Amer. Jour. Hyg.*, 27:493-497, 1938.

19. Berlin, B. S.: Gross Physical Properties of Emulsified Influenza Virus Vaccines and the Adjuvant Response. *Jour. Immun.*, 85:81-89, 1960.

PETROSITIS.*†

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This paper is a review of literature concerning inflammatory involvement of the petrous bone. Since the advent of antibiotics has markedly reduced the incidence of petrositis, its signs and symptoms are not readily recognized by many of us who entered otolaryngology in recent years. The anatomy, clinical findings and pathology of this condition have been reviewed and a case presented which illustrates many of these signs and symptoms.

In 1935 Kopetzky and Almour[1] reported ten cases of petrositis proven at surgery or autopsy. As these cases were handled before the advent of antibiotic therapy, cure depended upon surgical technique. They stressed the importance of doing a radical mastoidectomy in searching for the fistulous tract to the petrous bone. The majority of their cases had a fistula opening in or near the Eustachian tube orifice. These would have been missed, if only a simple mastoidectomy had been done. In their experience hearing loss was marked with only a simple mastoidectomy due to granulation tissue and destruction by the infection; little value was found in preserving the remaining hearing at the price of compromising the increased safety factor of a complete investigation of the middle ear and mastoid for fistulous tracts to the petrous bone. Adequate drainage of the petrous apex usually resulted in a cure, except in the case of a brain abscess formation. They also took exception to a subdural route to the petrous tip unless a subdural abscess was present. This approach opens a new route for infection to progress and does not give dependent drainage.

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In 1935 Eves[2] gave an excellent review of acute suppuration of the petrous pyramid. He points out the following anatomic relations associated with the symptomatology of petrositis: The acute cases of suppuration of the petrous pyramid occur most frequently in pneumatized mastoid bones. Extensively pneumatized temporal bones present cellular development extending into the zygoma, the squama, the occipital bone, the floor of the middle ear, around the mouth of the Eustachian canal well down into the anterior wall of the mastoid tip and into the petrosal pyramid. He reports that Carmack,[3] in a dissection study of 94 petrous pyramids in adults, found 32 per cent pneumatized. Myerson, Rubin and Gilbert[4] examined 200 temporal bones, both before and after removal from the cadavers and, by gross section, found only 11 per cent pneumatized petrous tips. This low percentage is largely due to the 23 specimens from infants. An interesting finding in their series is that 54 temporal bones from Negroes showed three times the percentage of cellular petrous pyramids as did those in white subjects.

The most generally recognized cellular pathways leading into the petrous pyramid are those described by Kopetzky[5]: *1.* From the tympanic space above or behind the superior semicircular canal, following the posterosuperior surface of the petrous pyramid into the tip; *2.* From the peritubal cells into the pyramid; *3.* From the peritubal cells by way of the carotid canal into the tip.

The great superficial petrosal nerve which, according to Piersol,[6] in all probability carries sensory as well as motor fibers, arises from the facial nerve in its canal and comes to the surface of the petrous pyramid through the hiatus fallopii. It then passes, usually in a groove on the surface of the petrous pyramid outside of the dura for a distance of 8 to 10 mm., before disappearing under the Gasserian ganglion to reach the foramen lacerum. Here it joins the deep petrosal nerve to form the Vidian nerve, thence through the Vidian canal in the sphenoid bone. The ascending orbital branch of the Vidian nerve passes through the sphenomaxillary foramen to supply the periosteum of the orbit and posterior ethmoid cells.

The fifth cranial nerve reaches the surface of the petrous bone through an opening in the dura after leaving the lateral side of the pons. The sensory roots form the Gasserian ganglion which is enclosed in Meckel's space, a delamination of dura lying in a slight depression on the apex of the petrous bone; internally, the Gasserian ganglion comes in relation with the cavernous sinus and internal carotid artery. Of the three branches given off from the Gasserian ganglion, we are concerned only with the first or ophthalmic branch. This branch is more firmly bound down by the dura in its longer course within Meckel's space than either of the other two, subjecting it to greater tension when irritation of the Gasserian ganglion occurs; the ophthalmic nerve passes upward and forward in the external wall of the cavernous sinus for about 25 mm., thence through the sphenoid fissure to supply the lacrimal gland, the upper eyelid and the skin around the external canthus.

The sixth cranial nerve is exclusively motor in function, supplying the external rectus muscle of the eye. Leaving the lower surface of the pons, it pierces the dura over the sphenoid bone, turns forward and passes between the apex of the petrous bone and posterior clinoid process of the sphenoid bone. A strong, fibrous bundle connecting these two bony prominences forms Dorello's canal. The superficial petrosal sinus as well as the abducent nerve passes through the canal. Passing forward and upward to the orbit, the abducens supplies the external rectus muscle.

Pain is the most characteristic and constant symptom of petrositis. It is located in, back of, and around the eye, in the temporoparietal region, sometimes reflected to the occiput of the affected side. This peculiar type of eye and associated pain persists and becomes more intensified until free drainage is afforded either by surgical means, by a large fistulous opening formed by necrosis into the middle ear, or rupture of the cortical surface of the petrous apex. Owing to the position of the great superficial petrosal nerve in a groove in the petrosal cortex, outside the dura, and 10 mm. nearer the origin of the infection, it seems logical to believe that the earlier pains come from its irritation. As the infection ex-

tends further into the apex it reaches the cells underlying the Gasserian ganglion and irritation of the ophthalmic nerve takes place.

Paralysis of the external rectus muscle is a relatively rare involvement in established cases of suppuration. It occurs only when the suppuration reaches the very tip cells of the bone involving the sixth nerve.

Profant[7] in 1931 also described the anatomy of the petrous bone in great detail. He brings out the following eight points:

1. Ossification of the labyrinth commences in the fifth fetal month at five main centers; by the end of the sixth fetal month the individual centers are more or less fused.

2. Until the seventh fetal month the petrous bone consists only of the labyrinth, tympanum and antrum. There is no petrous tip; hence the cochlea is at the apex.

3. At birth the petrous tip consists of rudimentary projections from the cochlea. The carotid canal is still a groove, and the internal auditory meatus merely an orifice. The antrum-epitympanic space has its adult characteristics. There are no petrous cells, but the molding of the future antrum-epitympanic route is seen in the new bone deposit extending outward to the zygoma and inward above the labyrinth.

4. At birth the labyrinth is practically the same size as in the adult, about 19 mm. The adult type of petrous bone, therefore, is the result of the molding of osseous deposits and structures about the fetal labyrinth, and it is reached between the fourth and fifth year.

5. The hypotympanic space also has adult characteristics at birth. The hypotympanic route of petrous cells is affected by the ossification of the adjacent carotid canal and jugular fossa below the labyrinth. The osseous space between the canal, fossa and labyrinth is the channel to the tip.

6. From a histologic standpoint the petrous cell is the same as the mastoid cell: a cavity lined with a thin mucoperiosteum. The development is coincident with and analogous to the development of the mastoid cell. The pneumatization of the

adult petrous bone depends on the amount and distribution of the bone deposit on the fetal labyrinth, as well as on the degree of transformation of this deposit into cells by the penetrating activity of the mucous membrane from the tympanum. This transformation is a normal process and not the result of an inflammation.

7. The dissections of the adult petrous bones show three types of bone formation: *a.* compact or sclerotic, *b.* spongy or diploic, and *c.* cellular. In a single specimen one, two or all three types may be present.

8. Because of the extreme variability of the pneumatization, no one case report, autopsy, or even examination of the specimen, can reveal the entire possible involvements of the petrous bone.

Pennybacker[8] in 1944 reported three cases of extensive cholesteatoma of the petrous bone. All three presented with slowly progressive facial paralysis, loss of taste and of hearing. They used a subdural approach to the cholesteatoma and felt it gave them good exposure, since each of cholesteatomas had eroded the superior surface of the petrous bone. In each case the X-rays were fairly diagnostic. It was not stated whether all the cholesteatoma had been removed. All cases were operated upon in the three years prior to the report, so little time had elapsed to allow for recurrence of the cholesteatoma.

Rambo[9] in 1957 presented a case of petrositis in a five-year-old girl. This girl had a simple mastoidectomy with continued discharge; followed by a radical mastoidectomy with an exploration of the petrous tip, again with return of discharge; finally a repeat exploration of the petrous tip, this time finding an abscess at the very apex. The patient's symptoms cleared following this procedure.

Niho *et al.*[10] give a detailed description of the anatomy of the petrous tip. They discuss the triad of Gradenigo, consisting of paralysis of the abducens nerve, neuralgia of the trigeminal nerve and acute otitis media. They point out that anatomically there is no abducens or trigeminal nerve run-

ning through the petrous bone; therefore, the involvement of these two nerves suggests extension of the disease beyond the petrous bone. They stress the importance of diagnosing and treating petrositis before it reaches this stage. Eberling[11] in 1952 reported a case of petrositis that responded to intensive antibiotic therapy. This patient had been treated for a bilateral otitis media with two injections of 600,000 units of procaine penicillin. Three weeks later she suddenly became unconscious and had convulsions. Spinal fluid showed no organisms, an elevated protein and one mononuclear cell. The patient had complete restoration of normal function.

Simpson[12] presented an interesting case of a 58-year-old man with an extensive petrous cholesteatoma. He had a history of an acute process with a twisted face 28 years previously, and continued to have a facial tic. In recent months his physician had noted a drooping of the right side of the mouth, and progressive deafness, without abnormal findings of the ear drum. A few months before the present illness he had had an acute vertiginous seizure. On examination he had trigismus, right facial palsy, no nystagmus, and some purulent discharge in the right external auditory canal (ear drums poorly seen). The spinal fluid was purulent. The patient expired, and on post mortem the petrous apex was completely replaced by cholesteatoma. There was no connection found between this cholesteatoma and the middle ear.

Missura[13] in 1959 reported two cases of petrositis. It was pointed out that with present antibiotic therapy the only symptom of Gradenigo's triad that still is consistently present is the pulsating headaches radiating into the eyes. In one of his cases the patient had had several previous mastoidectomies and at that time had a draining post-auricular fistula; however, his middle ear and hearing were essentially normal. With antibiotic therapy the infectious process in the middle ear is cleared, but a low-grade osteitis of the petrous bone may persist. Later this petrositis may flare up without subsequent involvement of the middle ear. Following a mastoidectomy, the middle ear may be sealed off from the mastoid and petrous cells, and so its function may not be affected by

later episodes of petrositis or mastoiditis. Of course, if the peritubal cells are involved, then the middle ear will also suppurate.

Hibler[14] also in 1958 presented a case in which the ear drum appeared normal, but an empyema of the apex of the petrous pyramid was present. This condition was extremely rare before antibiotics were given, and is usually caused by an inadequate antibiotic treatment of a preceding otitis media. In this case the otitis media was treated with penicillin and appeared to heal in two weeks. Three months later, deafness and vestibular elimination of the formerly diseased ear occurred so that a tumor of the acoustic nerve or of the cerebellopontine angle was assumed. Trephination by the neurosurgeon uncovered a pyramid apex empyema, which was operated upon from the ear three weeks later, and healed.

Jungmayr[15] in 1957 presented an interesting case showing an unusual cause of cholesteatoma. After a longitudinal fracture of the petrous pyramid, strangulation of the skin of the auditory meatus occurred. A peripheral tear of the tympanic membrane closed spontaneously. The strangulated epithelium proliferated at first like a tumor under the skin of the ceiling of the auditory meatus, and a dry cholesteatoma developed along the fracture fissure into the depth of the epitympanic recess, where it reached the anterior surface of the pyramid. It was removed by a radical operation. This case showed that with good pneumatization, a healthy auditory meatus, and a healthy tympanic membrane, the formation of a cholesteatoma is possible without secondary inflammation and merely strangulation of epithelium.

Csillag[16] in 1954 discusses in great detail the trigeminal pain associated with petrositis. He divides this pain into two categories: *1.* early pain; *2.* late pain. The early pain is found with otitis media and is relieved by a paracentesis or mastoidectomy. He theorizes this pain is due to venous congestion caused by the inflammation, creating pressure on the Gasserian ganglion. The late pain is due to actual inflammation and destruction of the apex of the petrous pyramid directly involving the Gasserian ganglion. This is not relieved without

a decompression of the petrous pyramid. Between these two periods of pain the lateral part of the petrous pyramid may be destroyed without pain. He also points out that trigeminal neuralgia is a much more consistent symptom than an abducens nerve palsy. Trigeminal pain is typical in disease of the anterosuperior pyramid face. The complex of symptoms originating at the glossopharyngeal, vagus, accessory, and hypoglossal nerves corresponds, when fully developed, to the nerve symptoms of a process in the pyramid advancing in the direction of the inferior pyramid face.

Schmidt[17] in 1952 reported a case of total destruction of the petrous pyramid in a 34-year-old man. The patient was asymptomatic except for some loss of hearing until an episode of meningitis. His case is similar to the one presented here. This patient demonstrates the delayed diagnosis of petrous bone involvement.

This 49-year-old male was found at 7:45 p.m. on November 28, 1929, in his apartment with a right temporal gunshot wound. The bullet entered the anterior portion of the right meatus, and there was bleeding from both the right ear and both nostrils. A right peripheral facial paralysis was noted. Skull films taken at that time showed metallic fragments lying anterior to the auditory meatus, extending down the postnasal region and over toward the left mandible. The right ear subsequently suppurated, and on December 13, 1929, a right simple mastoidectomy was done. No operative note was given, so the details of this procedure are not available. The patient was discharged from the hospital on January 9, 1930, with the ear still discharging. On March 20, 1930, a large bullet fragment was removed from the soft palate on the left in the Outpatient Clinic. During the two years following the injury the patient had slow partial return of function of the VIIth nerve; however, the ear continued to discharge. Mastoid films taken on May 20, 1931, showed a sclerotic right mastoid with a small fragment of lead above the ear canal, but quite deep. The left mastoid was read as normal. The patient did quite well then until 1942 when he developed an allergic rhinitis. His ear continued to discharge,

but otherwise gave him no difficulty. Sinus films taken at this time were read as normal paranasal sinuses.

In 1946 the patient first developed severe intermittent frontal and occipital headaches. Sinuses were again clear on sinus films, and these headaches gradually receded. On August 5, 1960, the patient developed excruciatingly severe headaches, associated with cerebrospinal fluid rhinorrhea. On August 19, 1960, the patient was admitted to the hospital where a

Fig. 1. Mastoid film taken while in hospital, 1960, showing destruction of the medial half of the right petrous bone.

lumbar puncture showed a white blood cell count of 2500 and cultured streptococcus and staphylococcus. The patient responded to large doses of chloromycetin, ilosone and penicillin. Within ten days the cerebrospinal fluid was normal. A carotid angiogram taken during this hospital admission was read as normal. The patient was discharged on oral penicillin. Mastoid films taken while in the hospital showed destruction of the medial half of the right petrous bone (see Fig. 1). Located within this radiolucent area were the lead fragments

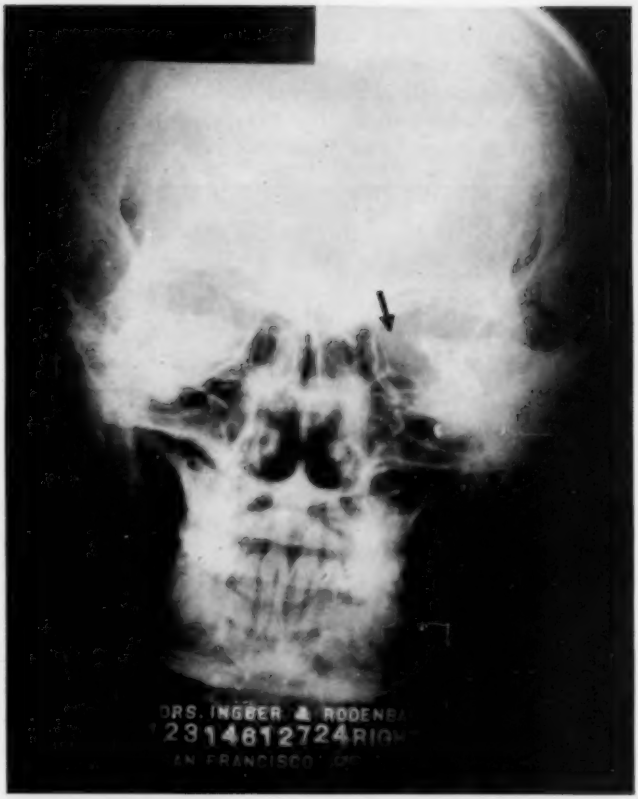

Fig. 2. Anterior view. Sinus film taken in 1946 showing petrous apex involvement medial to the lead fragments.

mentioned on earlier films. It was assumed that the lead fragments had carried in epithelial debris to form a cholesteatoma, or infection had travelled the path of the fragments to cause a suppurative petrositis; however, on the sinus films taken in 1946 (see Figs. 2 and 3), petrous apex involvement medial to the lead fragments was demonstrated. Even on the 1942 sinus films (see Fig. 4) the beginning involvement of the petrous apex is noted. In these films the radiolucency does not involve the area of the lead fragments, so these can

hardly be condemned as the cause of difficulty. When seen on September 30, 1960, the patient's right ear was dry. A cerebrospinal fluid rhinorrhea was present. A partial abducens palsy was noted on the right. It was decided that the source of meningitis could be either from the area of the cerebrospinal fluid rhinorrhea leak or from the dura over the petrous bone.

Fig. 3. Waters' view. Sinus film taken in 1946 showing petrous apex involvement medial to the lead fragments.

On October 18, 1960, an exploration of the right petrous bone was done. Through an endaural incision the squamous bone was removed above the tegman tympani and the dura elevated over the petrous bone. A lead fragment was removed from the superior surface of the petrous bone. The fragment did not appear to erode the dura. Medial and inferior to this fragment a large pocket of cholesteatomatous material was found, extending to the midline of the skull. The internal

carotid artery was seen pulsating within this cavity. The cholesteatomatous material was removed with suction, but it was impossible to remove the matrix. A drain was placed in the cavity and the incision closed. Pathology reported the material in the cavity to be keratin. Postoperatively the patient had no increased facial weakness. The patient did

Fig. 4. Waters' view. Sinus film taken in 1942 showing beginning involvement of the petrous apex.

have a profuse cerebrospinal fluid aural discharge and a right lateral rectus palsy was noted. Over a period of four days the cerebrospinal fluid aural discharge stopped, and the headache disappeared. Ten days postoperatively the cerebrospinal fluid rhinorrhea returned. One month postoperatively the patient developed meningitis which cleared rapidly with ap-

propriate antibiotic therapy. At that time the cerebrospinal fluid rhinorrhea stopped, and has not returned. At the present time the patient has no ear discharge, cerebrospinal fluid rhinorrhea or headaches. There is still some residual right VIth nerve palsy.

This case presents the triad of Gradenigo's syndrome over a prolonged period of time, leaving the etiology of cholesteatoma in this case still unanswered.

SUMMARY.

The literature on petrositis has been reviewed. While this has become an uncommon entity, it still occurs and may increase as more antibiotic resistant micro-organisms are manifested. Surgery is still an important factor to be considered in the treatment of this disease.

BIBLIOGRAPHY.

1. Kopetzky, S. J., and Almour, R.: Report of Ten Cases of Suppuration in the Petrosal Pyramid. *Ann. Otol., Rhinol. and Laryngol.*, 44:59-89, 1935.

2. Eves, C.: Acute Suppuration of the Petrous Pyramid. *Ann. Otol., Rhinol. and Laryngol.*, 44:97-109, 1935.

3. Carmack, J. W.: Discussion of Reference 25, p. 645. *Arch. Otolaryngol.*, Nov., 1934.

4. Myerson, M. C., Rubin, H., and Gilbert, G.: Anatomic Studies of the Petrous Portion of the Temporal Bone. *Arch. Otolaryngol.*, p. 195, Aug., 1934.

5. Kopetzky, S. J.: Problems Concerned with Empyema of the Petrous Apex. *Arch. Otolaryngol.*, 18:47-61, 1933.

6. Piersol, G. A.: Human Anatomy, Seventh Edition: Sixth Nerve, p. 1240.

7. Profant, H. J.: Gradenigo's Syndrome. *Arch. Otolaryngol.*, 13:3: 347-378, March, 1931.

8. Pennybacker, J.: Cholesteatoma of the Petrous Bone. *Brit. Jour. Surg.*, 32:125, 75-78, July, 1944.

9. Rambo, J. H. T., and Petti, G. H.: Petrous Apicitis. *Arch. Otolaryngol.*, 65:5, 523-524, May, 1957.

10. Niho, S., Niho, M., Yoshio, K., Iwatake, T., and Takahata, N.: Petrositis and Osteomyelitis Temporalis. *Yokohama Med. Bull.*, 5:2:125-138, May, 1954.

11. Eberling, E. W.: Acute Petrositis and Mastoiditis in a Patient Presenting Certain Unusual Diagnostic and Therapeutic Problems. *Mississippi Doctor*, 30:141-142, Oct., 1952.

12. Simpson, D. A.: "Cholesteatoma" of the Petrous Apex. *Jour. Laryngol. and Otol.*, 71:548-553, 1957.

13. Missura, T.: Neure Aspekte in Diagnose und Therapie der Pyramidenzellenneilerusgene. *Prac. Oto-Rhino-Laryngol.* (Basel), 21:1:49-55, Jan., 1959.

14. Hibler, N.: Pyramidenspitzenempyem bei Ausgeheiltem Mittelohr. *Monats. fur Ohrenheilkunde und Laryngorhinologie* (Wien), 92:4:205-208, Aug., 1958.

15. Jungmayr, H.: Cholesteatomentstehung nach Felsenbeinfraktur. *Zeits. fur Laryngologie, Rhinologie, Otologie und ihre Grenzgebiete* (Stuttgart), 36:6:365-368, June, 1957.

16. Csillag, A.: Die Petrositis Kennzeichnenden Trigeminusschinerzer. *Monats. fur Ohrenheilkunde und Laryngorhinologie* (Wien), 88:2:176-180, April-June, 1954.

17. Schmidt, M.: Cholesteatom mit Zerstorung der Ganzen Felsenbeinpyramide. *Prac. Oto-Rhino-Laryngol.* (Basel), 14:4:210-213, May, 1952.

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OSTEOCHONDROMA OF THE MANDIBULAR CORONOID PROCESS.*†

A Report of One Case and a Review of the Literature.

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Progressive restriction of mandibular motion resulting from the impingement of an enlarging coronoid process against the inner surface of the zygomatic arch was first reported in 1943 by Shackleford and Brown,[1] who described the restoration of normal jaw mobility in two youths after excision of expanded coronoid processes; in both cases, and in a subsequent case reported by the same authors in 1949,[3] the surgical specimens were reported to be osteochondromas. Nine additional cases of this unusual condition have since been reported by other authors.[2,4-10]

ANATOMY.

The coronoid process of the mandible is a thin, triangular eminence, which is flattened from side to side and varies in shape and size. The lateral surface of the process is smooth, and affords insertion to the temporalis and to the masseter; the medial surface provides the greater part of the area of insertion of the temporalis. The more important lateral relations of the coronoid process are to the zygomatic arch, the masseter, and the regional branches of the facial nerve.

The zygomatic branches of the VIIth cranial nerve pass anteriorly along the zygomatic arch, roughly parallel to the Frankfort plane, to supply the orbicularis oculi and to join with fibers of the lacrimal nerve and the zygomaticofacial

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Author	Date	Age	Sex	Location	Mandibular Excursion Pre-Op.	Approach	Remarks
Shackleford	1943	15	M	Left	12.5 mm.	External	Progressive Facial Swelling,
and Brown	1943	19	M	Right	10.0 mm.	"	3 Yrs. Erosion of Zygoma
Brant	1943	37	F	Right	25.0 mm.	External	Crepitation on Movement
Shackleford and Brown	1949	38	M	Right	Limited	External	Crepitation on Movement
Brailsford	1952	24	M	Left	12.5 mm.	Not Described	Limited Motion Was Only Complaint
Holmes	1956	30	M	Bilateral	7.0 mm.	Not Described	2 Stage Procedure with 2 Week
	1956	36	M	Left	8.14 mm.	"	Interval
Van Ziles and Johnson	1957	30	M	Bilateral	7.0 mm.	External	Eustachian Obstruction; 2 Stage Procedure with 1 Week Interval
Levine, Chessen, and McCarthy	1957	73	F	Left	6.0 mm.	Not Described	30 Yrs. Duration; Paper-Thin Zygomatic Arch
Dingman and Natvig	1957	27	M	Right	8.0 mm.	External	40 Lbs. Weight Loss
Antoni, Brown, and Johnson	1958	29	M	Left	No Limitation	Intraoral	Local Anesthesia; Intraoral Mass with Facial Asymmetry
Shira and Lister	1958	14	M	Bilateral	8.0 mm.	Submandibular, External, Subperiosteal	One Stage
Lebo	1961	18	M	Left	14.0 mm.	External	See Report

Fig. 1. Tabulation of selected data from the published case reports.

branch of the maxillary nerve. In the external approach to the coronoid process, it is important to identify these branches of the facial nerve so that they may be protected from injury.

PATHOLOGY.

Usually occurring in patients between ten and 25 years of age, and constituting the largest group of benign tumors

Fig. 2. A section through surgical specimen at the osseocartilaginous junction showing well-preserved trabecular pattern and marrow spaces. Note the regional variations of the cellularity in the cartilage and in the connective tissue.

arising from pre-cartilaginous skeletal connective tissue, the osteochondromas, or exostoses, are generally situated near the ends of long bones, forming osseous outgrowths capped by thin layers of expanding cartilage. The osseous pedicle arises directly from the cortex; there is no intervening stratum of abnormal bone. Geschickter considers these benign tumors, which he classifies with the potentially malignant chondromas and with the chondromyxosarcomas, to be exaggerations of normal bony protuberances intended as points of fixation for tendons.

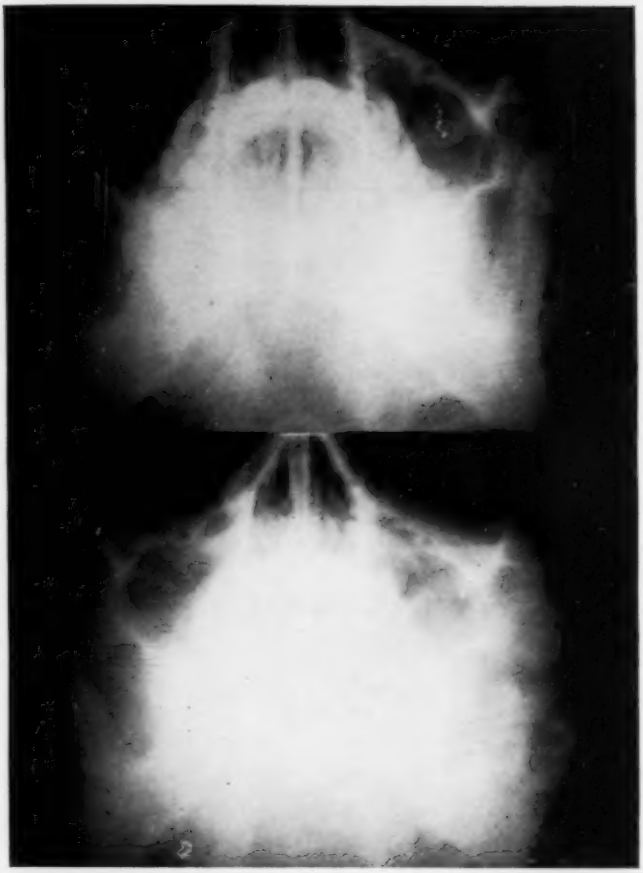

Fig. 3. Above: Preoperative X-ray of R.A. showing marked hypertrophy of the left coronoid process. Below: Postoperative view of same patient in identical projection.

The most superficial layer of the tumor is an hyalinized connective tissue containing islands of cellular connective tissue in which groups of small, spindle-shaped nuclei are syncytially arranged. The next layer is hyaline cartilage of the adult type, containing a few fetal islands; the deeper portions of this cartilage are undergoing calcification and are

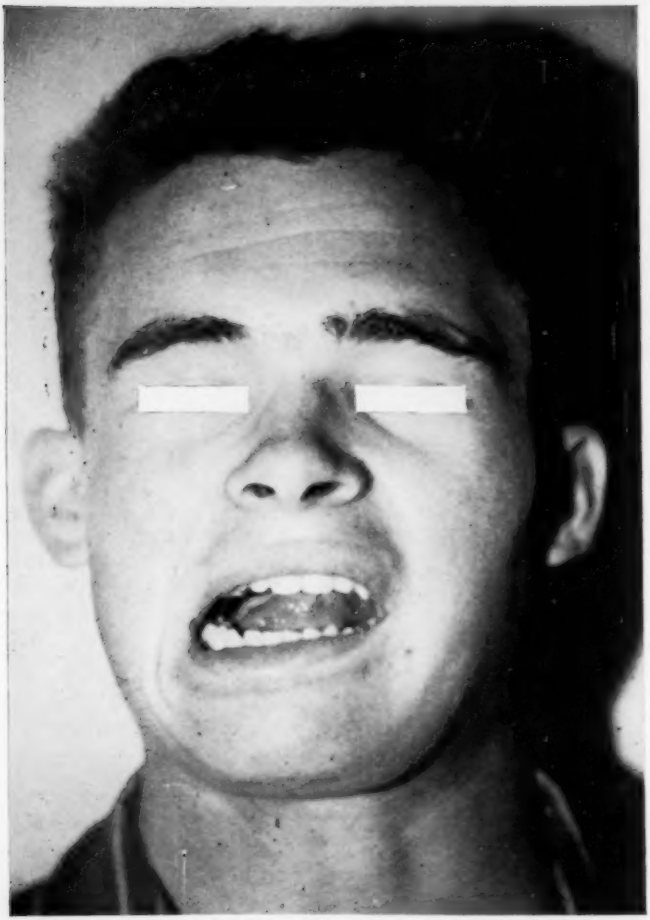

Fig. 4. Preoperative photograph of R.A. showing maximum intermaxillary opening.

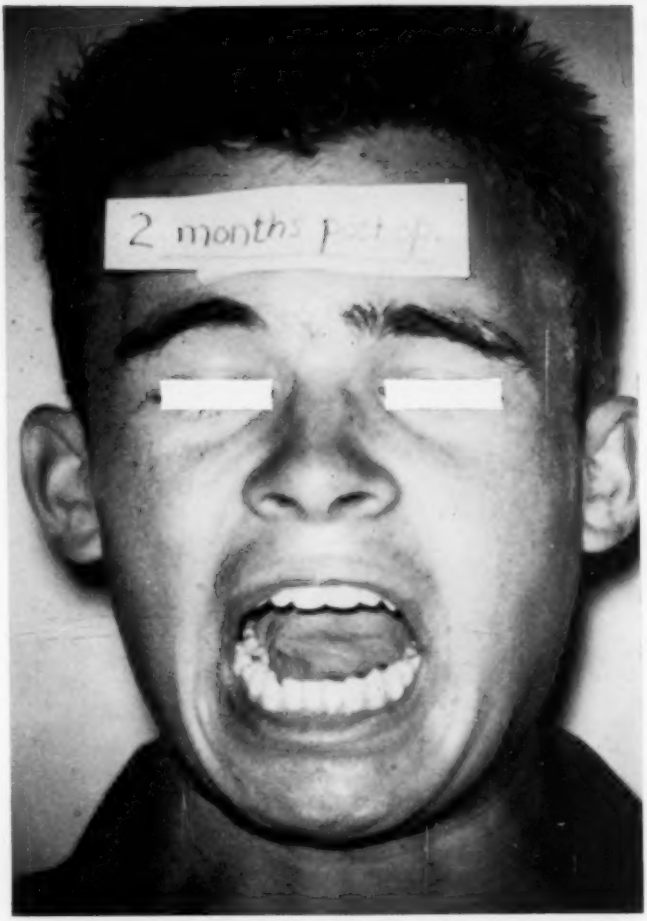

Fig. 5. Postoperative photograph of R.A. showing maximum intermaxillary opening.

in contact with laminated spicules of cancellous bone surrounded by a generous amount of fatty marrow.

CLINICAL FEATURES.

Of the 13 reported cases of osteochondroma of the mandibular coronoid process, ten or 76.9 per cent were unilateral. Only two, or 15.4 per cent, occurred in females. Excepting

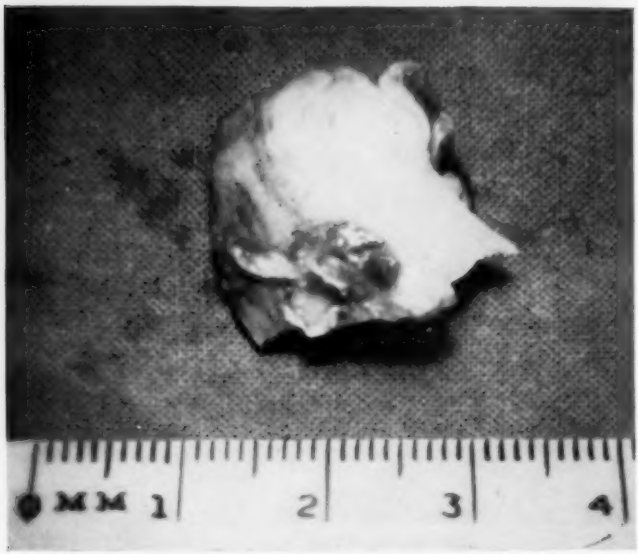

Fig. 6. The surgical specimen in this case.

the case involving the 73-year-old female,[7] the mean and the median ages in this series coincide at 26 years. Although in some of these cases the tumor had eroded the zygomatic arch and presented as a unilateral facial swelling, all patients retained some mandibular motion, the minimum reported preoperative intermaxillary opening being 7.0 mm.[5,6] In all cases, the progressive diminution of the range of mandibular motion was insidious and painless.

SURGICAL APPROACHES.

Of the reported cases in which the methods of surgical treatment were described, all but two were approached through horizontal incisions made directly over the zygomatic arch. In one case,[9] the involved coronoid process was excised through an intraoral approach. Shira and Lister[10] reported a one-stage procedure in which both coronoid processes were removed through external, submandibular, subperiosteal approaches.

CASE REPORT.

R. A., an 18-year-old male student, first examined on June 9, 1959, complained of slowly increasing loss of mouth-opening ability which he first noticed five years previously. This restriction, which was not anteceded by injury, was unaccompanied by pain, swelling, or crepitation. The remainder of the history was noncontributory.

The initial physical examination was negative except for limitation of vertical mandibular excursion to a maximum of 14.0 mm. No facial asymmetry was discernible, and there was no swelling in either zygomatic area. X-ray examination demonstrated marked enlargement of the left coronoid process. A diagnosis of osteochondroma of the left mandibular coronoid process was made.

On July 15, 1959, under general anesthesia, the anterior portion of the left zygomatic arch was exposed through a horizontal incision after identification of the regional branches of the facial nerve. Adequate exposure was obtained by the removal of a 12 mm. segment of the arch with a Gigli saw. The base of the coronoid process was then transected with a dental burr, and elevation of the periosteum permitted delivery of the process. There followed an immediate and dramatic increase in the range of passive mandibular movement.

The postoperative course was uncomplicated. Following a period of mild trismus, the patient became able to separate his upper and lower central incisors a distance of 29 mm. without discomfort.

SUMMARY.

A case of unilateral osteochondroma of the mandibular coronoid process occurring in an 18-year-old male is presented. In this unusual location, this common benign tumor produces progressive loss of the range of mandibular motion. The clinical features and management of 12 previously reported similar cases are described.

BIBLIOGRAPHY.

1. Shackleford, R. T., and Brown, W. H.: Osteochondroma of the Coronoid Process of the Mandible. *Surg., Gynec. and Obst.*, 77:51, 1943.

2. Brant, K.: Deformation Congenitale de l'Apophyse Coronoide du Maxillaire Inferieur. *Acta Orthop. Scandinav.*, 14:219, 1943.

3. Shackleford, R. T., and Brown, W. H.: Restricted Jaw Motion Due to Osteochondroma of the Coronoid Process. *Jour. Bone and Joint Surg.*, 31A:107, 1949.

4. Brailsford, J. F.: An Unusual Osteochondroma from the Coronoid Process of the Mandible. *Brit. Jour. Radiol.*, 25:555, 1952.

5. Holmes, F. H.: Mandibular Block by Osteochondroma of the Coronoid Processes. *Radiology*, 67:578, 1956.

6. Van Zile, W. N., and Johnson, W. B.: Bilateral Coronoid Process Exostoses Simulating Partial Ankylosis of the Temporomandibular Joint: Report of Case. *Jour. Oral Surg.*, 15:72, 1957.

7. Levine, M. H., Chessen, J., and McCarthy, W. D.: Osteochondroma of the Coronoid Process of the Mandible: Report of a Case and Review of the Literature. *New Eng. Jour. Med.*, 257:374, 1957.

8. Dingman, R. O., and Natvig, P.: Reduced Mandibular Motion Due to Osteochondroma of the Coronoid Process of the Mandible. *Amer. Jour. Surg.*, 94:907, 1957.

9. Antoni, A. A., Brown, A., and Johnson, J. H.: Osteochondroma of Coronoid Process of Mandible: Report of Case. *Jour. Oral Surg.*, 16:514, 1958.

10. Shira, R. B., and Lister, R. L.: Limited Mandibular Movements Due to Osteochondroma of the Coronoid Process. *Jour. Oral Surg.*, 16:183, 1958.

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AMERICAN LARYNGOLOGICAL ASSOCIATION AWARDS.

The DeRoaldes Award of the American Laryngological Association was presented to Dr. Frederick T. Hill, Waterville, Maine, in recognition of his many contributions to the specialty of otolaryngology and his services to the medical profession.

The Newcomb Award was presented posthumously to Dr. Chevalier Lawrence Jackson in recognition of his outstanding work in the field of Bronchoesophagology and for his untiring devotion to the furtherance of cordial relations with the medical fraternity of South America.

BELL'S PALSY.

A Ten-Year Review of Cases at the University of California Medical Center.*†

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and
WILLIAM M. HAMBLEY, M.D.,

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Doctor Robert C. Martin's keenly intelligent and well-informed opinions have exercised a considerable influence on VIIth nerve study and surgery for many years, at the University of California Medical Center. A review of idiopathic facial nerve paralysis cases covering the period 1950-1961, seen and treated, in the various clinics of the University should, therefore, provide some recognition of Doctor Martin's influence.

This study is not intended as a scholarly review of the literature on the subject of Bell's palsy nor even a detailed study of diagnostic and therapeutic measures. Both of these areas have been well covered by many previous authors including Martin,[1] Bunnell,[2] Moor,[3] Dalton,[4] Harris,[5] Rothendler,[6] Swan,[7] Cawthorne,[8] and many, many others. A very excellent bibliography and review of the problem may be found in the fine new volume Die Chirurgie des Nervus Facialis by Prof. Adolf Miehlke published in 1960 by Urban and Schwarzenberg in Germany.

During the ten years covered by this study there were 51 patients seen with an admission diagnosis of peripheral facial nerve paralysis.

Table I indicates the distribution of final diagnoses in

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*Read at the Scientific Program of Otorhinolaryngology in Honor of Robert C. Martin, M.D., University of California School of Medicine, San Francisco, Calif., April 15, 1961.

†From the Department of Otorhinolaryngology, University of California School of Medicine, San Francisco 22, Calif.

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these patients. Forty-one were confirmed as being Bell's palsy. The pathology in these cases undoubtedly lies in the changes in the facial nerve as it passes through the fallopian canal in the temporal bone. Some have felt that the majority of cases develop their pathology at or near the stylomastoid foramen where there is an anatomical narrowing of the facial nerve canal. Kettel[9] has also noted in some of his cases

TABLE I.

57 Patients—Peripheral Facial Paralysis.

Bell's Palsy	41 cases
Malignant neoplasms	2 "
Traumatic	6 "
Poliomyelitis	2 "

TABLE II.

Sex Incidence—41 Cases Bell's Palsy.

Male	Female
22	19

TABLE III.

Age Incidence.

0-10.....5	41-50.....7
11-20.....6	51-60.....6
21-30.....8	61-70.....1
31-40.....9	71-80.....1

TABLE IV.

Ear Involved.

Right	Left
17	25

actual softening and necrosis of bone in the area about the facial nerve canal.

Six of our cases were post-traumatic; two, in newborn infants with forceps deliveries; two post-mastoidectomy; one after skull fracture and one after gun-shot wound.

Tables II, III and IV give the sex, age and involved ear incidence respectively. It will be noted that there are 43

cases by age and involved ear incidence. One patient had a right Bell's palsy at the age of 12. Seventeen years later there was still a right lower facial weakness, and associated movements were present. At the age of 45 this patient suffered a left Bell's palsy. Six months later there was still no frontalis function on this side. Two years later some left facial weakness persisted.

Another patient, an infant, developed a right peripheral facial nerve paralysis at the age of two months. In two months this side had completely recovered; but in the interval, at three months of age, the infant developed a left facial nerve paralysis. At seven months of age, the left eye could be closed, but some residuals of the facial paralysis persisted. Three patients of the 41 were treated with steroids, the first, with Cortisone and the last two, with prednisone. The Cortisone-treated patient showed no improvement after three weeks. It must be pointed out that therapy was not begun until one week after the onset of the paralysis. The prednisone-treated patients began to respond at three and four days respectively after institution of therapy. In these two patients the prednisone was administered within three days of the onset of paralysis. The problem here is in determining what might be spontaneous return of function in a treated patient, since several of the untreated patients followed a course identical to steroid-treated cases.

Many of the patients showed some residual facial nerve weakness years after the onset of their paralyses. Because some follow-up examinations in our cases were only four to six months after onset, it was felt that recovery of function analysis would not be statistically valid.

In most large series of Bell's palsy cases 80 per cent recover spontaneously. It is the 20 per cent who may be left with serious permanent disability, for whom facial nerve "decompression" offers so much. Cawthorne[8] has stated that when the facial nerve paralysis is complete and return of function has not begun to take place at the end of one month, VIIth nerve exploration should be done. Doctor Martin has felt that at least one month should elapse after the onset of paralysis

before surgery is undertaken and that possibly two months would be the period of choice, since facial nerve decompression at this time could still result in return of function and since there would be little likelihood of spontaneous return after that length of time.

In this series of 41 Bell's palsy cases, four patients were operated upon. The first patient had a plastic left facial sling repair done in 1955 by Doctor Harry Blackfield and Doctor

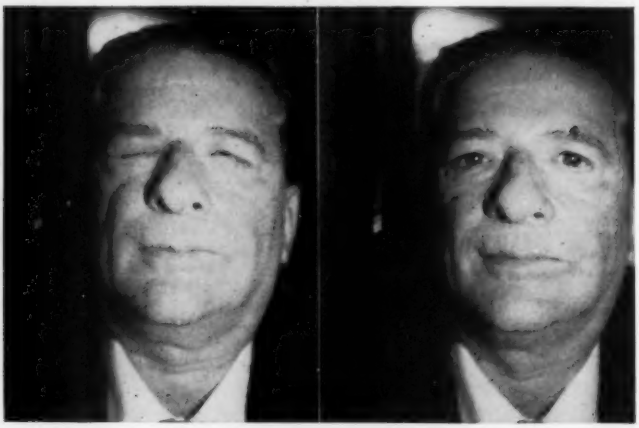

Fig. 1. Postoperative plastic left facial sling.

William Morris of the Plastic Surgery Department of the University of California. The excellent functional result of this procedure can be seen in photographs recently taken (see Fig. 1).

The other three surgical patients had facial nerve decompressions between 1958 and 1960, each one with excellent recovery as demonstrated in the photographs (see Figs. 2, 3, and 4).

Case 1. S. H., age 45 years, female, onset complete peripheral left facial nerve paralysis January 5, 1958. Facial nerve decompression April 17, 1958, because no return of function had occurred. By July 3, 1958, the patient was able to close her eye, and facial tone had returned, even

though, in this case, the nerve had been exposed only to within five millimeters of the stylomastoid foramen. In November, 1960, this patient developed a right facial paralysis but did not return to the clinic until April 3, 1961. At this time photographs (see Fig. 2) were taken. Right facial weakness was still present, left facial recovery was good with slight lower facial weakness persisting. Patient refused right facial nerve surgery at this time.

Case 2. E. E., age 73 years, female, onset complete right facial nerve paralysis December 1, 1959. No recovery of function and, therefore, surgery with complete facial nerve decompression, to stylomastoid foramen,

Fig. 2. Three years postoperative left facial nerve decompression. Untreated right Bell's palsy five months' duration.

was done on January 30, 1960. In one week slight movement began at right eyebrow and tone returned to right mouth. Photographs (see Fig. 3) were taken in April, 1961, and excellent return of function can be seen. Even the frontalis on the right was partly functioning.

Case 3. G. M., age 41, female, onset right paralysis May, 1960. No return of function in three months, and, therefore, a complete VIIth nerve decompression was done August 5, 1960. In five weeks, good muscle tone was present and voluntary movements of the lower face had returned. In four months from the date of surgery, there was a good return of function but with slight decrease in frontalis motion. The photographs (see Fig. 4) were taken at five months after surgery.

SUMMARY AND CONCLUSIONS.

Forty-one cases of Bell's palsy were seen at the University of California Clinic in the ten-year period beginning in 1950.

Fig. 3. Fifteen months' postoperative right facial nerve decompression. (Note frontalis motion.)

Fig. 4. Five months' postoperative right facial nerve decompression.

Medical therapy, including vitamins, steroids, etc., appeared to have no particular influence on the course of the paralysis. An appreciable number of patients showed no recovery of function or partial recovery only, many years after the onset of the paralysis. Three patients who had facial nerve decompression two to three months after onset of their paralysis, obtained excellent functional recovery.

It would appear advantageous to do a facial nerve decompression on any patient who has not shown at least the begin-

ning of a return of function after two months following a complete peripheral facial paralysis.

BIBLIOGRAPHY.

1. Martin, R. C.: Intratemporal Suture of the Facial Nerve. *Arch. Otolaryngol.*, 13:259, Feb., 1931.
2. Bunnell, S.: Surgical Repair of the Facial Nerve. *Arch. Otolaryngol.*, 25:235, March, 1937.
3. Moor, F. B.: Diagnosis and Treatment of Idiopathic Facial Paralysis. *Jour. A.M.A.*, 157, 18:1599, April 30, 1955.
4. Dalton, G. A.: Bell's Palsy: Prognosis and Treatment. *Brit. Med. Jour.*, 1:1765, June 11, 1960.
5. Harris, H. I.: Surgery for Facial Paralysis. *Plast. and Reconst. Surg.*, 13:193, 1954.
6. Rothendler, H. H.: Bell's Palsy Treated with Cortisone. *Amer. Jour. Med. Sc.*, 225:358, 1953.
7. Swan, D. M.: Stellate Block in Bell's Palsy. *Jour. A.M.A.*, 150:1, 32, Sept. 6, 1952.
8. Cawthorne, T.: The Pathology and Surgical Treatment of Bell's Palsy. *Proc. Royal Soc. Med.*, 44:565, Dec., 1950.
9. Kettel, K.: Bell's Palsy—Pathology and Surgery. *Arch. Otolaryngol.*, 46:427, 1947.

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CANCER OF THE LARYNX.*†

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INTRODUCTION.

This report is based upon our experiences in the care of 243 patients with malignancies of the larynx and hypopharynx. Ninety-eight per cent of the cases had primary squamous cell carcinomas while the remaining two per cent had malignancies of other cell types. The diagnosis in each was based on microscopic examinations of tissue from biopsy specimens.

The anatomical classification of the primary lesion in each patient is shown in Table I. In 127 the lesion arose on the true vocal cord or cords. Hoarseness is an early symptom of cordal cancer, yet in spite of this, 57 per cent of the patients did not report for examination until the disease was advanced, often beyond the limits of the cords. The remaining 116 patients had primary lesions that arose in anatomical sites other than the true vocal cords. They were invariably examined in late stages, no doubt due to the vagueness of early symptoms. Thirty-one per cent of this group of patients showed palpable cervical lymph node metastases when they were first examined while only two per cent of the patients with cordal cancer showed like metastases. On the other hand 66 per cent of the patients with pyriform sinus primary lesions showed palpable nodes, Table I.

When this study was completed, 137 patients were alive, Table II. For the whole series of 243 cases, this represents

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an over-all survival rate of 57 per cent, Table III. According to the anatomical site of the primary lesion, 72 per cent of those living had cordal cancer, whereas only 38 per cent of the patients with primary lesions "other than cordal" were living. Note that these percentages do not represent five-year survival rates.

Table I.

Anatomical Location and Stage of Lesions in 243 Cases.

BASED ON CLASSIFICATION BY INTERNATIONAL RADIOLOGICAL CONGRESS – COPENHAGEN, 1953.

			PALPABLE NODES		
	STAGE I	STAGE II	STAGE III	STAGE IV	
Cordal	55	69	2	1	= 127
Supraglottic		61	20	1	= 82
Pyriform sinus		6	11	1	= 18
Subglottic		1	1		= 2
Pharyngeal		1	5		= 6
"Other primaries"					= 4
"Multiple primaries"					= 4
				TOTAL	243

Cases Treated Over Five Years Ago (138).

One hundred and thirty-eight patients were treated five or more years ago. Table IV indicates that 50 per cent of them were alive when this study was completed. It is quite evident that the highest percentage (71 per cent) of patients living had primary cordal cancer while only 28 per cent of cases with non-cordal cancer survived. The anatomical site of origin of the primary lesion apparently has much prognostic significance when viewed from this aspect.

Table V indicates that seven cases died of cordal cancer, ten of other causes and there was no follow-up in three. Forty-five cases died of cancer when the primary lesion arose in non-cordal sites and four died of other causes. This seems to indicate that cancer deaths occurred in the latter group before the toll of intercurrent disease became apparent.

Table 2.

Summary of 243 Cases Treated (245 lesions)

LOCATION OF LESION	NO.	LIVING	DIED CANCER	DIED OTHER CAUSES	NO FOLLOWUP
Cordal	127	92	10	20	5
Supraglottic	82	38	41	3	0
Pyriform sinus	18	3	13	2	0
Subglottic	2	0	2	0	0
Pharyngeal	6	1	4	1	0
*Multiple primaries	5	2	2	0	0
*Other than sq.cell & cordal	5	1	3	0	0
TOTAL LESIONS	245	137	75	26	5

*(2 cases cross-indexed.)

243 CASES

Table VI shows the cases treated by X-ray therapy, combined X-ray therapy and surgery and by surgery alone. Note the survival rates with each form of treatment.

Table VII denotes that 38 patients were treated with X-ray therapy alone and that 34 per cent survived. Ten of the 13 patients living had cordal malignancies.

Table VIII shows an additional 22 cases that received X-ray therapy initially and had subsequent surgery due to unarrested cancer or chondronecrosis of the thyroid cartilage. Most of these cases have been reported elsewhere.[1] Eighteen

of these cases were laryngectomized, 12 for cancer, five for chondronecrosis and the remaining case for inability to swallow and repeated aspirations (this patient had had X-ray therapy followed by transhyoid epiglottectomy). The four remaining cases had more extensive surgery, including block dissections of the neck for unarrested cancer. All four died

Table 3.

Survival to Date of 243 Cases.
(1 to 13 years)

LOCATION OF LESION	NO.	LIVING	
Cordal	127	92	(72%)
Supraglottic	82	38	(47%)
Pyriform sinus	18	3	(16%)
Subglottic	2	0	(0%)
Pharyngeal	6	1	(16%)
Multiple primaries	4	2	(50%)
Other than sq.cell ℓ cordal	4	1	(25%)
	243	137	

Alive at completion of study 57% (All forms of treatment)

of cancer. Fifty per cent of the 22 patients survived five years or longer. It is pointed out, however, that three of the four patients that died of other causes within the five-year period, had laryngectomies for chondronecrosis and "no cancer."

Table IX indicates the number of patients (76) treated surgically. Since the cancer was too far advanced in two cases, no treatment was possible. Again it is evident that the highest percentage (70 per cent) of patients surviving had cordal primary lesions as opposed to 43 per cent of the cases with non-cordal cancer.

Table X indicates combined groups of cases namely, those treated by surgery alone (76 cases) and those treated by surgery following X-ray therapy (22 cases). Seventy per cent of the patients survived five years when the operative procedure deemed necessary to cure the patient was laryngectomy or a lesser operation, while only 37 per cent survived when more extensive operative procedures were necessary.

Table 4.

Cases Treated 5+ Years Ago (138)

(All types of treatment. 2 cases untreatable)

LOCATION OF LESION	NO.	LIVING 5 TO 13 YEARS		
Cordal	69	49	(71%)	
Supraglottic	46	17	(36%)	
Pyriform sinus	13	2	(15%)	
Subglottic	2	0	(0%)	28%
Pharyngeal	4	0	(0%)	
Multiple primaries	3	1	(33%)	
Other than sq.cell + cordal	1	0	(0%)	
	138	69		
5+ yrs. survival		50%		

(7 lived 5+ years, died of intercurrent disease;
3 died with no cancer chondronecrosis; 3, no follow-up.)

Cases Treated Less Than Five Years Ago (105).

One hundred and five cases have been treated within the last five years, Table XI. Already 23 patients have died of the primary cancer, 12 of "other causes" and there was no follow-up in two cases. No further comment will be made here regarding this group of patients, but rather they will be discussed later in this report particularly in reference to certain recent trends in treatment.

COMMENT.

During the past 20 years, the treatment of primary malignancies of the larynx and its adnexa has undergone many changes. The most important are the recognition of the place of radical surgery in the treatment of cervical lymph node metastases, the merits of X-ray therapy, and, although seemingly paradoxical, the recognition and the part played by conservative surgical treatment.

Table 5.

Summary of 138 Cases Treated 5+ Years Ago.
(139 lesions; 2 cases untreatable.)

LOCATION OF LESION	NO.	LIVING		DIED PRIMARY CANCER	DIED OTHER CAUSES	NO FOLLOWUP
Cordal	69	49	71%	7	10	3
Supraglottic	46	17		27	2	0
Pyriform sinus	13	2		9	2	0
Subglottic	2	0		2	0	0
Pharyngeal	4	0	28%	4	0	0
Multiple primaries	3	1		2	0	0
*Other than sq. cell & cordal	2	0		1	0	0
	139	69		52	14	3

*Case "other than squamous cell" cross-indexed with "pyriform sinus".

The presence of resectable cervical lymph node metastases or the possible presence of microscopical metastases in patients with advanced primary malignancies in these areas dictates radical neck dissection in continuity with excision of the primary lesion. The observation of cancer-bearing lymph nodes by microscopical tissue examinations in the excised specimen should alert the surgeon to the consideration of contralateral radical neck dissection even though the pri-

mary lesion may be unilateral. This dictum applies in those patients where the primary lesion is located near the midline or crosses it. This is applicable to cases with non-cordal lesions for the most part, but is also applicable in those few cases with advanced bilateral cordal lesions.

Factors other than anatomical location of the primary lesion and palpable cervical lymph nodes which aid the surgeon in planning treatment are: size of the primary lesion, edema,

Table 6.

Types of Treatment in 138 Patients Surviving Over 5 Years.

	NO. CASES	LIVED 5+ YEARS	
X-ray therapy alone	38	13	(34%)
X-ray therapy + surgery	22	11	(50%)
Surgery (all types)	76	45	(60%)
Too far advanced for therapy	2	0	(0%)
	138	69	or 50%

ulceration, exophytic growth, impairment of function and malignant cell differentiation; microscopic examination of the excised tissue will often give the surgeon clues as to adequacy of excision. This applies to primary as well as to cervical metastatic disease. Microscopic malignant cells invading perichondrium and cartilage demand immediate radical neck dissection. This was shown in one patient who developed inoperable cervical lymph node metastases seven years after laryngectomy. Upon reviewing this patient's original laryngectomy specimen, microscopic nests of cancer cells were

present in the perichondrium of the thyroid cartilage. The patient's original primary lesion arose on the true vocal cord.

If there is doubt in the mind of the surgeon as to the magnitude of the initial surgery, he should perhaps favor radical excision. The policy of "watchful waiting" for cervical lymph node metastases to occur following laryngectomy has been discouraging. The five-year survival rate in these patients

Table 7.

38 Cases Treated by X-ray Therapy Alone and Surviving Over 5 Years.

LOCATION OF LESION	NO.	LIVED	DIED PRIMARY CANCER	DIED OTHER CAUSES	NO FOLLOWUP
Cordal	13	10	1	0	2
Supraglottic	18	2	16	0	0
Pyriform sinus	7	1	6	0	0
	38	13	23	0	2

34% SURVIVAL RATE

76% "cordal" lived;
12% other than cordal.

is not over 25 per cent even though block dissection of the neck is performed later; whereas, if block dissection had been done in continuity with laryngectomy in these patients, the survival rate could no doubt have been improved.

In certain patients, the primary lesion may be in such a position (pharyngeal wall, lateral surface of the epiglottis, etc.) that partial pharyngectomy and/or partial laryngectomy can be done. Usually in such instances, ipsilateral block dissection of the neck is done in continuity.

In the whole series of 243 cases, X-ray therapy was used

as an initial treatment in 95 cases. The X-ray therapy was given in 16 different offices or clinics located over a widespread geographical area. It is true that patients with small true vocal cord primary squamous cell carcinomas can be treated by X-ray therapy as successfully as by conservative surgery. This series of cases bears this out. In addition, in most instances the voice is preserved after X-ray therapy. It appears that more advanced cordal malignancies are better

Table 8.

Initial X-ray Therapy Followed by Surgery in 22 Cases Treated 5+ Years Ago.

LOCATION OF LESION	NO.	LIVED	DIED PRIMARY CANCER	DIED OTHER CAUSES	NO FOLLOWUP
Cordal	14	9	1	4	0
Surpraglottic	5	2	3	0	0
Subglottic	1	0	1	0	0
Pharygeal	2	0	2	0	0
	22	11	7	4	0

50% SURVIVAL RATE

treated by surgery. A high percentage of the patients who received initial X-ray therapy and required surgery later, had more advanced primary cordal cancer. Malignancies that arise "other than the true vocal cord" are better treated by surgery than by X-ray therapy (see Tables VII, VIII and IX); however, some patients with highly anaplastic primary lesions may have a rather dismal outlook changed for the better by the use of initial X-ray therapy followed by surgery. The complications of X-ray therapy and surgery are at a minimum. No case of radiation perichondritis has been seen during the

past five years, nor has there been a death attributable to surgery in the whole series of patients.

It is deemed better to make the decision for X-ray therapy or surgery at the outset and not hopefully expect one form of treatment to supplement or replace the other in case of failure. The X-ray therapist should be as carefully chosen as the surgeon. Close teamwork between the two may bring many benefits to the patient.

Table 9.

Initial Treatment—Surgery (all types) 5+ Years Ago.

LOCATION OF LESION	NO.	LIVING		DIED PRIMARY CANCER	DIED OTHER CAUSES	NO FOLLOWUP
Cordal	41	29	70%	4	7 (3 after 5 yrs)	1
Supraglottic	25	14		9	2	0
Pyriform sinus	5	1	43%	2	2 (after 5 yrs)	0
Subglottic	1	0		1	0	0
Pharyngeal	4	1		3	0	0
	76	45		19	11	1
*Too advanced for treatment	2					
	78					

67% SURVIVAL RATE

45+5=50 CASES

*(1 case pyriform sinus, 1 supraglottic.)

Patients with primary malignancies of the pyriform sinus and the subglottic area have a poor prognosis with any form of treatment. Cervical and upper mediastinal lymph node metastases occur early. Two patients with primary subglottic squamous cell carcinomas succumbed soon after treatment (one of these was treated by X-ray therapy followed by laryngectomy, the other had laryngectomy and block dissection of the neck in continuity). Seventeen cases of pyriform sinus cancer were treatable; one was untreatable. Of those

treated seven had X-ray therapy and ten surgery. Fifteen patients have died. One case treated by X-ray therapy is living 11 years post-radiation. He had a primary submucosal lymphoepithelioma of the pyriform sinus. Of those treated by surgery, one is living less than one year following surgery, and one is living five years. Of the 15 patients who died, two lived longer than five years and died of other causes.

Table 10.

Surgery - 98 Cases.
(76 surgery; 22 initial X-ray and later surgery.)

		NO. PTS. LIVING 5+ YEARS		
Peroral endoscopic	2	2	70%	3 lived less than 5 years, no cancer chondronecrosis
Laryngofissure	12	9		
Hemilaryngect.+skin gr.	2	2		
Laryngectomy	45	29		
Miscellaneous group: Laryng.+bl.dissec. in cont., Laryng.+bilat.bl.dis.in stages, Laryng.+later block or blocks, Block dissection.	37	14	37%	4 lived 5+ years, died of other causes.
	98	56		

65% SURVIVAL RATE

Unfortunately, X-ray therapy or surgery used alone or in combination cannot be considered a panacea in the treatment of patients with malignancies in these areas (see Table VI). In general, the most conservative treatment should be chosen for any individual patient compatible with eradicating the cancer and preserving function. Ideally, peroral endoscopic removal of the malignancy would be the goal in every case; but, of course, this is impractical and impossible.

In studying the cases (105) treated within the last five years, the following trends have been evident:

1. Patients with cordal malignancies suitable for conservative surgical excision, have been referred in greater numbers to the X-ray therapist for treatment.

2. Hemilaryngectomy has been the operation of choice in an increasing number of suitable cases.

3. Block dissection of the neck in continuity with laryngectomy and/or partial laryngectomy and pharyngectomy is

Table II.

Summary of 105 Cases Treated Less than 5 Years Ago
(106 lesions)

LOCATION OF LESION	NO.	LIVING	DIED CANCER	DIED OTHER CAUSES	NO FOLLOWUP
Cordal	58	43	3	10	2
Supraglottic	36	21	14	1	0
Pyriform sinus	5	1	4	0	0
Pharyngeal	2	1	0	1	0
*Multiple primaries	2	1	0	0	0
Other than sq. cell & cordal	3	1	2	0	0
	106	68	23	12	2
		105 cases			

*1 case "multiple primaries" cross-indexed with "supraglottic".

being performed in a greater number of cases than previously. Conversely, fewer laryngectomies are being performed as a lone surgical procedure.

4. Contralateral block dissection of the neck is being performed with increased frequency in certain patients. The reasons for this were pointed out in a previous paragraph.

"Other Than Primary Squamous Cell Malignancies" (Five Cases).

In the group of 243 patients, five had "other than squamous cell primary malignancies": four of the larynx and one of the pyriform sinus. These cases are unusual and are noted as follows:

Case 1. J. V., male, age 31, had a primary myxosarcoma of the left aryepiglottic fold. This was resected by lateral pharyngectomy July 22, 1947. One year later he died of widespread pulmonary metastases.

Case 2. R. B., male, age 55, had a primary submucosal lymphoepithelioma of the right pyriform sinus. He is living and well 11 years following X-ray therapy.

Case 3. C. H., male, age 63, had a primary pseudosarcoma of the left aryepiglottic fold extending into the medial wall of the left pyriform sinus. On June 16, 1958, a laryngectomy and left radical neck dissection were done in continuity. At present he is living and well two and one-half years after surgery.

Case 4. D. B., male, age 63, had primary lymphosarcoma of the right aryepiglottic fold with bilateral cervical lymph node involvement. Following X-ray therapy in 1959 the laryngeal lesion regressed. Subsequently he died of lymphosarcomatous liver involvement.

Case 5. C. S., female, age 55, had a primary melanoma of the left aryepiglottic fold. Since the larynx is an extremely rare site of origin of melanoma, a subsequent biopsy was done. The two biopsy specimens were diagnosed as melanoma. A thorough search for other possible primary sources of this lesion proved fruitless. The patient refused all therapy. Two years after the original examination she is dying of cervical lymph node metastases.

Multiple Squamous Cell Primary Lesions (Four Cases).

Four of the 243 patients had multiple squamous cell carcinomas in the larynx and adjacent pharyngeal areas. The site of origin of the primary lesions precluded the possibility of direct extension. Still another patient not included in this group of four patients showed multiple primary squamous cell carcinomas of the tracheal wall extending to the seventh ring. This latter patient had an advanced primary lesion of the true vocal cord which had been treated originally by X-ray therapy. The tracheal lesions were found at surgery.

Cases with multiple pharyngeal and laryngeal primary lesions present many difficulties in treatment. A case in point is R. C., male, age 57. In 1955 he developed a primary squamous cell carcinoma of the right lateral pharyngeal wall. On Oct. 18, 1955, a right partial pharyngectomy and block dissection of the neck were done in continuity. In 1959 a second primary pharyngeal lesion developed higher on the

same side of the pharynx. On Feb. 10, 1959, in order to remove this lesion, a portion of the right side of the mandible and base of the tongue were excised in continuity with the pharyngeal wall. Still later he developed a large fungating lesion of the left aryepiglottic fold and left side of the epiglottis. On July 28, 1959, a laryngectomy and left radical dissection of the neck were done. The pharyngeal and laryngeal mucosa in the excised specimen in this latter instance showed areas of multiple primaries by microscopic tissue examination. The patient has been living five and one-half years since the original surgery.

The other three patients in this group have not had such widespread new primary cancers, and consequently have, for the most part, been treated with a combination of X-ray therapy and/or local excisions.

Submucosal Primary Lesions (Four Cases).

Submucosal primary lesions developed in four of the 243 patients. These cases present many problems in diagnosis.[2] In two patients with submucosal primary lesions of the false vocal cord, diagnosis was finally made by thyrotomy, removal of tissue and frozen section microscopic tissue examination. Several attempts at biopsy by the direct method had failed to reveal the primary lesion in either case. In one the presenting complaint was a mass in the left side of the neck which by biopsy excision proved to be squamous cell carcinoma. Laryngectomy and left radical neck dissection in continuity were performed July 28, 1948. Two years later lymph nodes developed in the right side of the neck and a right block dissection was done on July 24, 1950. Five years after the date of the original surgery, the patient fell, fracturing his skull. He died subsequently of a massive subdural hematoma. Autopsy revealed no evidence of cancer. The second patient had a submucosal primary lesion of the left false vocal cord which was diagnosed by thyrotomy and frozen section. Laryngectomy was done one month later. The patient died of cervical metastases within 18 months.

The other two patients in this group had submucosal pri-

mary lesions of the pyriform sinus. One had squamous cell carcinoma; the other, lymphoepithelioma. The latter was treated with X-ray therapy and is living 11 years post-radiation (noted above). The other died within two years of cervical metastases in spite of block dissection and radiation therapy.

Distant Primary Malignancies (13 Cases).

In the whole series of 243 cases, 13 patients had distinct primary malignancies as shown in the following list:

Lung malignancies	7 cases
Tonsil	4 cases
Prostate	1 case
Testicle	1 case

There was little question of reciprocal metastases in these cases, for in most instances the laryngeal malignancy was of an entirely different cell type.

SUMMARY.

1. A series of 243 cases of primary malignancies of the larynx and hypopharynx are reviewed. Ninety-eight per cent of the primary lesions were squamous cell in origin.

2. In 52 per cent of the patients the primary lesion originated on the true vocal cords, and only 2 per cent showed palpable cervical lymph node metastases, while in the group of patients who had "primary lesions other than the true vocal cord," 31 per cent showed palpable cervical lymph nodes.

3. Of the 138 cases treated over five years ago, 50 per cent have survived, without evidence of cancer, for five or more years. Of 38 patients treated by X-ray alone, 34 per cent survived and of 22 patients treated by X-ray and surgery, 50 per cent survived.

In 76 patients treated by surgery alone, 67 per cent survived. (An additional two patients were not treated because of far advanced cancer.)

In 98 surgical patients (76 plus 22) the five-year plus

survival rate was 70 per cent with patients who had primary cordal lesions, and 37 per cent in cases with "other than primary cordal lesions." The over-all survival rate in these 98 cases was 65 per cent.

4. In the treatment of malignancies of the larynx and hypopharynx, the recognition of the following are important.

a. Radical surgery in relation to cervical lymph node metastases.

b. The merits of X-ray therapy in early cordal cancer.

c. The place of conservative surgery in eradicating primary cordal cancer.

5. Based on the treatment of 105 cases within the past five years the present trends of treatment were noted.

6. Five unusual cases of primary malignancy of the larynx and hypopharynx other than squamous cell type are noted.

7. Four cases of multiple primary squamous cell carcinomas of the larynx and pharynx are presented.

8. The difficulties in diagnosis are noted in four patients with submucosal primary lesions.

9. Thirteen cases with distinct distant primary malignancies are reported.

CONCLUSIONS.

Squamous cell carcinoma of the larynx and hypopharynx is still a formidable disease. In patients with early primary cordal cancer, survival rates indicate that surgery and X-ray therapy are just about equally effective in treatment. Surgery should be performed in cases with more advanced primary cordal lesions.

In patients with primary squamous cell carcinomas arising in areas "other than the true vocal cord," surgery is the treatment of choice; however, in certain patients with highly anaplastic primary squamous cell malignancies, X-ray therapy may often be considered initially. At best this latter group

is difficult to treat, and the patient's outlook for survival is little changed even with subsequent surgery.

Although 51 per cent of the 243 cases had primary true vocal cord carcinomas, 57 per cent of them did not seek medical care until the malignancy was in an advanced stage. Apparently the symptom of hoarseness is still not the danger signal it should be.

BIBLIOGRAPHY.

1. Work, W. P.: Surgery of the Neck Following Radiation Therapy for Cancer. *Ann. Otol., Rhinol. and Laryngol.*, 68:393-410, 1959.

2. Work, W. P.: Primary Squamous Cell Carcinoma of the False Vocal Cord. *Calif. Med.*, 85:107-109, Aug., 1956.

VEIN GRAFT MYRINGOPLASTY.*†

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The purpose of this paper is two-fold: first, to report experiences with vein graft myringoplasty in a small series of cases in a general Ear, Nose and Throat practice; secondly, to re-emphasize that vein grafts appear to be a distinct forward step in reparative ear surgery.

Shea[1] performed the first closure of an eardrum perforation with a vein graft in December 1957, as reported in his article in the Archives of Otolaryngology in October 1960. He describes the basic technique, of using the operating microscope, of denuding the free edge of the perforation by means of fine, angle-tipped instruments, separating the outer squamous layer from the inner mucosal layer. The vein graft, previously trimmed of excess adventitia and slit lengthwise to make a flat piece, is tailored to fit the perforation. It is supported on pledgets of absorbable gelatin sponge (Gelfoam®), placed in the middle ear space, the intima inward.

Tabb[2] has designed a knife which facilitates the incising of the free edge of the perforation. In his article in THE LARYNGOSCOPE in March 1960, he stressed the denuding of the inner surface of the eardrum adjacent to the perforation, for a distance of a millimeter or two, to afford a bed for the graft; thus the graft, placed within the middle ear, overlaps the perforation. This technique was used in the cases here reported. In addition, the medial wall of the middle ear, seen through the perforation, was scarified to cause bleeding, for possible additional blood supply, as described by House.[3]

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The incision at the donor site, to obtain the segment of vein, has varied, sometimes made transversely across the vein, other times longitudinally, directly along the vein. While it would seem cosmetically better to follow the skin lines and make a transverse incision, the flexion and extension of the upper extremity joints seem to widen such an incision scar, so that at present the longitudinal incision is usually used.

Table I lists some of the details of the 11 patients making

TABLE I.

	Sex	Age	Ear and Length of Postop. Follow-up		Extent of Perforation	Operative Result
1. M.L.	F	46	Right	15 mo.	20%	**Closed secondarily with patching
			Left	5 mo.	95%	*Primary closure
2. L.E.	F	46	Left	15 mo.	50%	*Primary closure
3. G.N.	M	37	Left	14 mo.	25%	****Failure
4. P.F.	F	31	Right	13 mo.	25%	*Primary closure
5. R.N.	F	46	Left	13 mo.	25%	Failure
			Re-operation 6 mo.			***Closed
6. A.M.	M	46	Right	9 mo.	25%	**Primary closure, then tiny perforation closed by patching
			Left	3 mo.	20%	****Failure
7. H.V.	M	7	Left	10 mo.	40%	Failure
			Re-operation 6 mo.			***Closed
8. B.S.	F	12	Right	10 mo.	90%	*Primary closure
9. J.S.	M	12	Left	10 mo.	40%	*Primary closure
10. J.A.	M	16	Right	7 mo.	25%	*Primary closure
11. G.S.	F	35	Right	4 mo.	30%	*Primary closure

(Asterisks refer to Summary in Table II.)

up the series. These patients represent 13 ears that were operated upon for a total of 15 operations. The longest postoperative follow-up period, on the first case done, is 15 months (operated on January 14, 1960). The most recent case included in this series has been followed for four months.

Of the 13 ears operated upon, the primary operation completely closed the perforation (complete "take" of the graft), in seven cases (53 per cent). Two additional eardrums had tiny perforations postoperatively but closed completely after

patching-techniques were used. Addition of these two, to the seven primarily closed, gives a success result of 69 per cent (see Table II).

Two failures, in which the grafts simply atrophied, were done a second time, and both were completely successful. Two additional failures have not been re-operated.

The successes with re-operation may well be related to the better local conditions in the ear for reception of the graft, due to the primary surgery.

TABLE II.

Totals.

	No. of Ears	No. of Operations
*Primary complete closure	7	7
**Secondary closure with patching	2	2
***Failure but closed by reoperation	2	4
****Failures, not as yet reoperated	2	2
	13	15

Details of some of the cases are of interest.

The first vein graft myringoplasty in this series (Case 1) was on a "pseudo-eardrum," a patient who, three years previously, had a Type III tympanoplasty on the right ear, using post-auricular skin. A perforation, about 20 per cent of the "eardrum" developed; covering the perforation would bring the hearing up to serviceable levels. After using patching techniques, a split-thickness skin graft closure was tried and failed; then a pedicled skin-graft, from the mastoid bowl and ear canal, was used and failed. The December 1959 advance program of the Southern Section of the American Laryngological, Rhinological and Otological Society listed the preliminary report by H. G. Tabb on closure of tympanic membrane perforations by vein grafts, subsequently presented at the Section Meeting on January 16, 1960. By letter Doctor Tabb kindly forwarded the gist of his experience, and on January 14, 1960, the perforation in this tympanoplasty case was successfully closed by vein graft.

This same patient had had bilateral ear disease years ago, and also lacked most of the opposite (left) eardrum. Because of this, detail of the middle ear, such as the incudo-stapedial joint and round window niche, could be easily seen. This afforded a wonderful demonstration for medical students. Vein graft replacement of this eardrum, approximately 95 per cent absent, was done five months ago with primary complete success.

Case 2 was a perforation originally a sequel to a stapes mobilization procedure. At a subsequent Lempert fenestration, attempt was made to close the hole by plicating the edges together. When this failed, closure by post-auricular skin-graft was attempted; it too failed; therefore, vein graft myringoplasty was employed, and primary closure has now been observed for 15 months. Serviceable, unaided hearing was obtained. With all the procedures carried out in this patient, by the time of the vein graft the perforation involved about 50 per cent of the eardrum. Somewhat embarrassing, donor vein graft from the wrist area proved to be too small; a segment of a larger vein, farther up the arm was taken, and appeared adequate until placed in the middle ear; finally a vein-segment from the antecubital area proved adequate.

A vein-press has been proposed to thin, flatten, expand, and make vein-grafts easier to handle. Such a device might have obviated the need for extra vein-segments taken in this case. It is probably improper to express an opinion about this, without experience in its use, but it would seem obvious that some cells would be killed by such compression, and such handling would violate a basic surgical tenet.

While most of these operations were performed through the ear speculum in the ear canal, three required endaural incisions (Lempert incisions 1 and 2) with retraction of the external auditory meatus and a variable amount of funneling of the bone by dental drill, for good exposure of the perforation (Cases 3, 4 and 10). When this was contemplated, especially in children, general anesthesia was used; it was used electively in most of the patients in this series.

Footplate fixation suggesting otosclerosis was found in Case 5. Now that the eardrum is intact, fenestration of the oval window with polyethylene prosthesis is being considered to close her air-bone gap.

Case 6 might very properly be classified as a primary closure. Primary closure occurred, but six months later a tiny perforation developed; it closed readily with a single application of a patch. This perforation was a surprising development. Since so many months had passed, it was felt

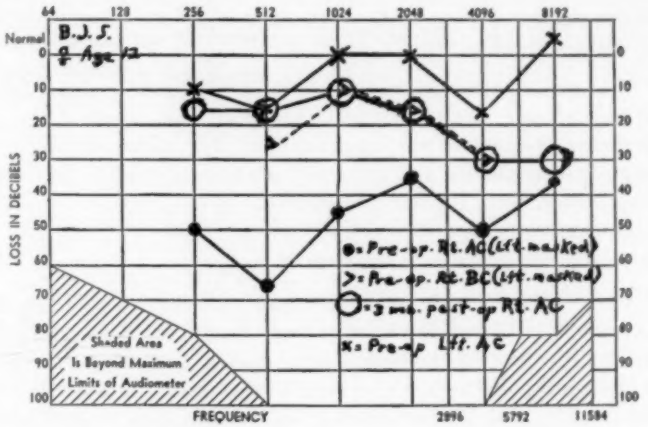

Fig. 1. Pre- and postoperative hearing tests in Case 5, vein-graft myringoplasty in 12-year-old girl, incus absent.

the closure was permanent. The patient did not have a cold or ear infection, but had noticed some "popping" of the ear in crossing a high hill by car.

Case 7, a seven-year-old boy with bilateral perforations and hearing loss, had a marked speech defect. In the six months since successful closure of the left perforation his speech has improved with the gain in hearing. He was very recently hospitalized for operation on his right ear (not tabulated); it was interesting to note the pediatric house-officer describe the left, previously operated, eardrum with

a curt, but accurate, "Left eardrum O.K." Certainly it is impressive that usually the vein-graft-repaired eardrum resembles the normal tympanic membrane.

Case 8, a 12-year-old girl, is of special interest because at surgery it was discovered that the incus was absent. Figure 1 illustrates the pertinent hearing curves, including the preoperative 40 to 20 db. air-bone gap. The intra-tympanic Gelfoam® was, therefore, carefully placed so that the vein graft still rested on the head of the stapes. Gelfoam® was also placed externally on the graft, pressing it against the stapes. The graft was *not* incised to fit on the head or neck of the stapes. Figure 1 illustrates the complete closure of the air-bone gap obtained.

Skin-graft myringoplasty had been completely successful on the left ear of Case 10, but vein-graft was used by choice to repair the second ear, also successfully. McLaurin, *et al.*,[4] recently emphasized the generally greater suitability of vein, rather than skin, for ear procedures.

COMMENT.

As mentioned, vein-graft-repaired eardrums resemble, to a surprising degree, the normal tympanic membrane. Several successful closures, however, have had a fibrous, pink spot in the center of the graft. Possibly this represents a fibrous band, containing blood vessels from the scarified promontory.

Vein graft myringoplasty as here presented has been of a general, basic type. At present all kinds of variations are being tried by otologists, with many ingenious techniques. With experience, some of these will prove particularly worth while. With such knowledge, however, the surgeon still will have to decide the best procedure(s) for the individual problem of a given case.

SUMMARY.

Vein graft myringoplasty, first performed by Shea, can be a very gratifying procedure for both the patient and surgeon. Results in a small series of 11 patients, by a fairly

basic technique, are reported. Further experience with many varied techniques now being tried will eventually prove the worth-while procedures, for application by the surgeon to the individual problem encountered.

BIBLIOGRAPHY.

1. Shea, John J., Jr.: Vein Graft Closure of Eardrum Perforations. *Arch. Otolaryngol.*, 72:445-447, Oct., 1960.

2. Tabb, Harold G.: Closure of Perforations of the Tympanic Membrane by Vein Grafts. The Laryngoscope, 70:271-286, March, 1960.

3. House, H. P.: Surgical Repair of the Perforated Eardrum. *Ann. Otol., Rhinol. and Laryngol.*, 62:1072-1082, Dec., 1953.

4. McLaurin, J. W., Raggio, T. P., and Tabb, H. G.: A Technique of Tympanoplasty. The Laryngoscope, 71:116-130, Feb., 1961.

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THE VEIN GRAFT IN RECONSTRUCTIVE EAR SURGERY.*†

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Recent interest in the reconstructive surgery of the ear drum has led to the widespread search for a satisfactory method of procedure and a suitable donor material. Among the latter, skin and vein have been the most popular. It is the purpose of this paper to lend support to those who advocate the use of vein grafts. I should like further to demonstrate that not only is the vein graft suitable for closing small tympanic membrane perforations but that it can also be successfully employed in reconstruction of a totally destroyed ear drum.

In comparing the use of skin and vein, in all fairness, it should be stated that in the use of skin there are certain advantages: first, the skin is usually available from the same operative field, either post auricular or from the canal wall; second, it is easier to prepare the graft bed from the presenting or squamous side of the drum rather than having to reach under it with a hook to prepare the mucosal or middle ear surface; third, it is easier to get proper approximation of the graft to the bed by packing it down with gelfoam than it is to build up a bed of gelfoam pledgets in the middle ear for the vein to lie upon.

With further reference to the use of skin, often the graft continues to shed epithelium for weeks, and occasionally months, before it eventually becomes dry. Skin takes a considerable, although varying, period of time before the graft assumes the physical appearance of a normal drum. Then

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too, we often find that after an apparently successful skin graft, at periods of time from a few months to over a year, a breakdown and slough of the graft may occur. Surgically induced cholesteatoma is another problem which makes the prospect of using skin less attractive.

Using vein as the graft material offers certain distinct advantages which I feel are significant. There is no epithelial debris and the ear is often dry as soon as the canal is cleansed postoperatively. A vein does not seem to have the same problems of nutrition that skin does. I surmise that this is partly due to the intima's receiving part of its nutriment from the blood within the vessel. This is closely matched by the intimal surface's being in contact with the gelfoam mass, almost invariably with some blood in it, supporting it from the middle ear side. Healing is often complete in a month and the graft quickly assumes the appearance of a normal drum. One exception, Case 5, required a cigarette paper patch to close a 2 mm. perforation in the center of the graft postoperatively; but, nonetheless, the portion that had taken primarily on this total reconstruction looked like normal tympanic membrane after one month. The paper patch was successful after two applications.

It has been my experience in this small series that if a vein graft is used as a primary procedure, the likelihood of a closure of the drum is very favorable, whereas if the vein is used to close a persistent perforation after a partially successful skin graft, this, at least in my hands, offers a greatly decreased likelihood of success. There have been no failures with the primary vein graft procedure in nine cases, whereas, in three cases where the vein was used as a secondary procedure, there has been uniform failure. In Case 8, although the perforation was closed, the patient noticed a severe drop in her hearing two days postoperatively.

Preoperatively it was insisted upon that the ear be absolutely dry for at least three months prior to surgery and that the patient have a history of being able to go through an upper respiratory infection without developing a draining ear. In the case of the ear that did drain with upper respira-

tory infections, local treatment to the ear was instituted with Chloromycetin-boric acid dusting powder, frequent cleansing, steroid-antibiotic drops and self-inflation of the Eustachian tube. This was combined with appropriate treatment of the nose and nasopharynx until the above-mentioned conditions could be met.

Postoperatively the patient kept a pledget of cotton in his external canal for two weeks during which time the ear was not disturbed. Cleansing of the ear was performed about the fourteenth day and an audiogram was made after one and four months. No postoperative Eustachian tube inflation was done.

It is my feeling that the term myringoplasty might well be abandoned and that all these procedures be called tympanoplasty of varying types. In every case I feel that the middle ear should be explored for unexpected pathology and to insure the continuity of the ossicular chain. This can still be done in most cases by the per-aural route, using a wide stapes type of flap and removing bone of the scutum. Despite the absence of clinical or X-ray findings, cholesteatoma and chronic infection must be ruled out positively and not merely by inference. If these conditions are found at exploration then the reconstruction must take a back seat to curing the disease. Continuity of the ossicular chain must be confirmed by direct inspection and observation of a transmitted motion. If the chain is interrupted, appropriate steps to improve hearing can be taken at the same time.

Case 1, Mr. J. W.: This 54-year-old white male had had bilateral tympanic membrane perforations since having scarlet fever as a child. There had been no drainage for many years previous. A kidney bean-shaped perforation was found on both sides. An audiogram revealed an equivalent SRT by bone of 0 on both sides, and by air, of 32 decibels on the right, and 42 decibels on the left. The patient underwent surgery on September 16, 1960. When inspecting the ossicular chain it was found that an otosclerotic process involved the footplate of the stapes with a thick, dense, white footplate. No attempt was made at attacking this problem at the same time.

The patient, when seen in the office on December 2, 1961, had an intact, movable, normal appearing left tympanic membrane. He still had an air-bone gap of 40 decibels which is felt to be due to the otosclerotic process. This will be approached at a later date.

The second perforation has subsequently been successfully repaired. The footplate was fixed, but thin. There was no arch of the stapes. *In*

situ footplate fracture was accomplished, and ossicular continuity was restored with a polyethylene strut. At three weeks postoperative the patient says he can hear, but no audiogram is available as yet.

Case 2, Mr. P. C.: This 39-year-old white male athletic instructor had a history of a perforated tympanic membrane on the right for many years and showed an air-bone gap of 50 decibels with normal bone thresholds. The opposite ear was within the limits of normal.

Surgery was undertaken on August 28, 1960, and was uneventful. The ossicular chain was found to be intact, and there was no evidence of disease in the epitympanic space.

Two weeks later on September 9, 1960, the perforation was found to be closed, and the patient reported his hearing was improved.

On September 27, the ear drum had the appearance of a naturally occurring ear drum, and there was no air-bone gap.

Case 3, Miss S. E.: This 14-year-old white female had a history of chronic tympanic membrane perforation on both sides since early childhood. She had previously had a left tympanoplasty with a skin graft done by me with a preoperative air-bone gap of 40 decibels.

Postoperatively the gap was narrowed to 15 decibels; however, just recently we have seen late desquamation of the graft, and I fear that its continued existence is in doubt. On the right there was an initial air bone gap of 35 decibels which disappeared on patching.

A vein graft procedure was performed on August 3, and by August 25, the ear had healed and the air bone gap was 10 decibels.

Case 4, Miss E. W.: This 8½-year-old white female had a history of persistent left tympanic membrane perforation following an acute otitis media associated with an upper respiratory infection several years previously. There had been no recent drainage. An audiogram showed an air-bone gap of 15 decibels.

A vein graft procedure was carried out on June 29, 1960, and on July 8, 1960, the patient reported she was hearing equally in both ears. Healing was progressing satisfactorily.

The patient did not reappear until August 25, at which time the air-bone gap had been closed, and the healing was complete.

Case 5, Sister M. A., OP: This 68-year-old white female had a history of chronic tympanic membrane perforation for over 20 years which had been dry since the acute episode. The equivalent SRT by bone was 30 decibels and by air 52 decibels in the involved ear.

Physical examination revealed a complete absence of the ear drum on the left although the annulus was present. A reconstructive procedure was undertaken on July 19, 1960, and was notable only for the fact that the patient experienced a prolonged anectine reaction and had to be ventilated artificially for four hours after the termination of the procedure.

By August 9, the operative site was found to be healing well, but a 2 to 3 mm. posterior inferior central perforation was found. This was patched with cigarette paper and the patient did not return in two weeks as instructed but rather appeared on September 21, at which time, the patch was found to have fallen off. The perforation persisted. The perforation was repatched and when seen on October 7, it was well healed. An audiogram at that time showed an equivalent SRT by air of 30 decibels, thus eliminating the air-bone gap.

Case 6, Mr. R. O.: This 17-year-old Mexican male had a history of

chronic bilateral ear drum perforations since he was a small boy. There had been no history of drainage within the past several years.

Physical examination revealed a kidney bean perforation on both sides with normal bone thresholds and an air-bone gap of 40 decibels on each side.

Reconstructive procedures were carried out bilaterally at two separate times using skin, and a satisfactory result was obtained on the left with closure of the air-bone gap. The procedure on the right ear showed a primary take in all aspects, but after a few months it was apparent that cholesteatomatous-like debris was being extruded antero-inferiorly. The patient was returned to surgery, and the cholesteatomatous matrix was removed and a vein graft was substituted. This showed no indication of taking and sloughed completely. The air-bone gap on the right is now 30 and the ear requires intermittent attention for purposes of hygiene.

Case 7, Mr. H. B.: This 58-year-old white male welder had his left tympanic membrane damaged by a spark in 1954. A previous attempt at reconstruction of the ear drum with skin by another surgeon was partially successful, and when he came to me the inferior one-third of the tympanic membrane was still missing.

An attempt at closing the remaining perforation was made on September 7, 1960, and a vein graft was used. At first it seemed as though the graft would take, but one month postoperative there had been a total slough of the graft despite denuding and scarifying the undersurface of the previous graft as well as the remaining annulus and the apparent absence of cholesteatoma.

Case 8, Mr. C. McL.: This 42-year-old white male welder incurred a traumatic perforation of his left ear drum from a welding spark in June of 1960, which did not respond to conservative therapy and which measured 5 to 6 mm. in diameter.

Surgical repair was performed on September 16, 1960, and by October 11, the drum was well healed, had a normal appearance, and the previously existing air-bone gap of 15 to 20 decibels was closed.

Case 9, Mrs. K. F.: This 55-year-old white female had a long standing attic perforation on the right. The preoperative air-bone gap was 15 decibels. Without giving all the details, the arch of the stapes and the incus were absent; therefore, the footplate was connected to the malleus by means of a polyethylene strut. In so doing the footplate was displaced into the labyrinth in part. The patient heard beautifully for two days postoperatively and then lost it all. She was dizzy for three months. She now has an air-bone gap of 50 decibels and perhaps a dead ear.

SUMMARY AND CONCLUSIONS.

The use of vein as a material for closing tympanic membrane perforations is discussed. Representative cases are presented. In all cases exploration of the epitympanic space and ossicular chain for defects in continuity and unsuspected disease must be made.

Since this article was submitted for publication, one of the cases referred to as a success has had a recurrence of the perforation.

384 Post Street.

PROBLEMS ENCOUNTERED IN TYMPANOPLASTIC SURGERY.*†

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Modern tympanoplasty surgery is only 12 to 15 years old. Wullstein[1] and Zollner,[2] *et al.*, reopened this chapter in Europe and, with modern surgical microscopy and antibiotics, were able to give new impetus to old concepts.

With the vast reservoir of less privileged individuals, economically and medically, the dearth of treatment during World War II, wartime disabilities, and the lack of antibiotics, there probably are many more chronic otologic problems in Europe than in this country.

In the United States, with Lempert's[3] pioneering work on fenestration and Rosen's[4] rediscovery of stapes mobilization, our interests had been directed primarily toward restoration of hearing loss in otosclerosis and perfecting techniques accordingly.

The economic resurgence and increased travel, both to and from Europe after World War II, facilitated the exchange of ideas and techniques, with the result that modern tympanoplastic surgery became more widespread in the United States. By the same token, our experience in fenestration and stapes surgery was felt in Europe and the rest of the world.

Stapes surgery *per se*, and its astonishingly good results over longer periods of time, further helped in the development of tympanoplastic surgery. Patients began to consult otologists not for eradication of infection of the mastoid and

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middle ear, but rather for correction and improvement of hearing.

Many patients experienced initial disappointment on learning that they were not candidates for stapes surgery; however, because of their renewed interest in the possibility of hearing again, they were more amenable to discussing tympanoplastic surgery.

It is surprising to discover that the draining, suppurating ear, *per se*, is often accepted by the patient and his family as a condition with which they are resigned to living; consequently, discussions on eradication of infection to prevent potential secondary serious complications—such as inter and intracranial spread—are frequently rebuffed. This is probably due to the common usage of antibiotics, locally and systemically, to control acute exacerbations of chronic otologic conditions, which lulls the patient into a sense of security—as false as it may be.

In many instances, the non-otologic specialist promulgates this; however, when the discussion is turned to possibilities of restoration of hearing, slight as they may be, interest is almost always evoked by the patient and his family. It is important for the otologist to recognize this fully and realize the responsibility he has to the patient and himself regarding the primary purpose of tympanoplastic surgery; that being primarily the total eradication of infection, and secondarily the preservation, maintenance and/or restoration of hearing.

The otologist must maintain his objectivity as much as possible, for tympanoplastic surgery is fraught with great technical difficulties and frequent poor results regarding restoration of hearing. A review of the literature will bear this out.

The type, degree and scope of preliminary audiometric determinations have been fully discussed in previous papers; therefore, we are assuming that the patient has been audiologically evaluated completely and competently prior to surgery so that an attainment potential has been established.

As in other procedures, before the surgeon approaches tympanoplastic surgery he must be well versed in the physiology and pathology of the temporal bone. He must have had adequate experience in surgery of the temporal bone and its environs, and he must be familiar with all the available equipment and instruments necessary to this procedure. A constant awareness of techniques being used by others must be maintained inasmuch as this is an evolutionary type of surgery, and one that will not remain static. The otologic surgeon must also be current on the world literature, participate in special work shops and courses, and avail himself of opportunities to observe the actual surgery itself as performed by other otologists.

Wullstein's classification of tympanoplasty outlines the five basic methods of converting the sound apparatus in the temporal bone to a working mechanism for conducting sound. Unfortunately, however, all tympanoplasties do not fall neatly into one of these categories and the findings at surgery sometimes necessitate a modification of one of the five types, or a combination of several of the types Wullstein has outlined. The decision the surgeon must make at this time will draw on his extensive personal experience and previous observations.

In a review of our cases of tympanoplastic surgery performed from October, 1958, to the present, including all types a la Wullstein (categories I through V), we have, in most instances, achieved our primary aim, namely, eradication of disease of the temporal bone. In cases where revisions have been performed due to infections, we have found with few exceptions that the infections have been due to recurrent cholesteatosis. Children have constituted the majority of our revisions, and in several cases it has been necessary to perform more than two procedures.

Our secondary aim of preservation and/or restoration of hearing has not been so satisfactory. We have successfully reoperated some of our patients who had no initial gain, while we have revised others several times with no improvement, or only slight improvement.

In assessing the cause of failure on an individual and collective basis, we have found that the failures fall into one of two categories: *1.* the patient was not a candidate for tympanoplastic surgery; or *2.* the patient was a candidate but failed to reach his predicated neural level or experienced further loss.

Patients who are not candidates for tympanoplastic surgery will evince the following contra-indications:

1. Diffuse Cholesteatosis.

This condition exists primarily in children. In spite of the benefit of microscopic surgical aids, the most competent otologic surgeon in these cases is unable to be absolutely positive that all cholesteatoma has been eradicated. Rather than bury potential cholesteatoma and infection, we feel that the most extensive radical mastoidectomy possible should be performed, and with the knowledge that the cavity and middle ear will epithelialize, no attempt should be made to close the middle ear space initially with a graft.

2. Bacterial Infections.

Certain bacterial infections which are resistant to antibiotics will persist in suppurating in spite of radical surgery to the temporal bone. Not all of the cells of the temporal bone can be exenterated, and covering up infection will result only in the loss of the graft as a minor consequence, and probable intracranial complications as a major consequence; particularly in the case of B Proteus pseudomonas and tuberculosis.

3. Tympanosclerosis.

This condition is characterized by deposits of large masses of hyalinized material in the submucosal layers of the tympanic cavity and mastoid, where ossicles are usually embedded. While attempts to remove localized areas may seem successful and results of tympanoplasty are successful, we have had occasion in one case to have the process actually involve the graft. Covering the middle ear and/or mastoid is, in essence, the same as covering a case of diffuse cholesteatosis.

4. Deficiency or Atrophy of Mucosa of the Middle Ear.

This applies to the patient with an adhesive thin drum plastered to the promontory which, at separation, reveals bare bone over most of the middle ear. Attempts at reconstructing a cavum minor with mucosal implants, *viz.*, segments of buccal mucosa, sinus lining, veins or plastic materials, have not been very successful.

5. Closed Eustachian Tube.

This condition cannot always be fully determined before surgery, and the factors of allergy, sinusitis, nasal obstruction, endocrine status, anatomical abnormalities, local infection and polypoid changes about the intratympanic orifice and pharyngeal orifice must first be evaluated. Attempts to correct these conditions when present must be made prior to surgery. Forced inflation of greater than 30 mm. pressure to aerate the middle ear will not succeed in maintaining tubal patency, and the surgery is doomed to failure.

6. Presence of Acute Infection.

Acute exacerbations of a chronic ear, or acute infection *per se* requiring extensive temporal bone surgery certainly should not have tympanoplastic surgery simultaneously. A secondary procedure may be performed at a later date.

On those patients who were by all indications good candidates but who failed to reach their predicated neural level or experienced further loss, we made the following observations:

1. Split Thickness Grafts.

In most instances it was only a matter of time before the graft had sloughed in part and gradually enlarged to lay bare the middle ear. Initially the results in improved hearing have been excellent but later deteriorated, necessitating a revision with a full thickness skin graft.

2. The Use of Polyethylene Struts.

The results of improvement in hearing in Type III over

Type IV tympanoplasty led us to use a polyethylene strut as a contact with a mobile footplate and the graft, when the stapedial crura were necrotic or destroyed. Initially, the results were most satisfactory, but in time the strut eroded through the graft; the erosion enlarged, there was suppuration, and the strut had to be removed with the graft. A Type IV tympanoplasty was then performed.

3. Manipulation of the Stapes and/or the Footplate.

In all instances we were dealing with an infected middle ear. The body had succeeded in building barriers against this infection. A fixed stapes due to otosclerosis, tympanosclerosis or inflammation is a tempting area to manipulate. A footplate buried in fibrous tissue is also a tempting area to manipulate, to test phase relationship with the round window. Manipulation which is too assiduous can result in opening the labyrinth and, as a lesser complication, cause a serous or circumscribed labyrinthitis. A major complication is the total loss of hearing. In stapes surgery with no infection, the number of cases resulting in total deafness have increased with more footplate manipulation. When infection is present the chances of permanent damage is greatly increased.

4. Lack of Mucosa of the Middle Ear.

This has been described previously.

5. Recurrent Cholesteatosis.

This has also been described previously.

6. Infection of the Cavity.

The otologist must develop scrupulous techniques for the after care of the cavity. We do all dressings in our office and none in the hospital. Patients on whom we operate—whether from out of town or clinic patients—must come to the office for postoperative care for a minimum of three weeks. This period is extended if the ear is not healed sufficiently at the end of that time. If secondary infection does develop, then all steps must be taken to eliminate the secondary infection entirely or the graft will sough in part or *in toto*.

7. Improper or Inadequate Follow-up on the Part of the Patient.

It is surprising that the occasional patient who is so desirous to hear will show a complete lack of wisdom in following instructions pertaining to postoperative care. He may miss appointments, not take the required medication, or fail to call when a complication develops. Some patients have gone swimming, put pieces of Kleenex or toilet tissue in the ear, cleansed their ears with match sticks and cotton, and ignored systemic illnesses as related to their ears. All attempts must be made to follow these patients and treat them adequately—in spite of themselves.

8. Thickening and De-Epithelialization of the Graft.

The full thickness graft gradually de-epithelializes and thins out, finally becoming quite elastic. In some instances, however, the graft will continue to de-epithelialize and slough its outer layers. The hearing result will be satisfactory, but the cavity will remain wet and must constantly be cleansed and kept dry. This is disturbing and must be watched to preclude secondary infection.

SUMMARY.

1. There has been a resurgence of tympanoplastic surgery since World War II. Although it is an old art, modern surgical microscopy and antibiotics have given a new impetus to this type of surgery. Whereas our aims were directed toward perfection of techniques for improvement of hearing in otosclerosis, the Europeans, namely Wullstein and Zollner, were the modern pioneers in tympanoplastic surgery.

2. We should be objective in our aims toward restoration of hearing in patients having chronic suppuration. We must realize our first responsibility is the eradication of infection in the temporal bone, and our secondary aim the preservation and restoration of hearing.

3. It is definitely established that some patients are not suitable candidates for tympanoplastic surgery, either as a primary or secondary procedure.

4. In patients who were suitable candidates for tympanoplastic surgery, but who failed to reach their predicated neural level or experienced further loss, reasons for failure must be ascertained.

BIBLIOGRAPHY.

1. WULLSTEIN, H.: Die Tympanoplastik als Gohoverbessernde Operation bei Otitis Media Chronica und ihr Resultate. *Proc. Fifth Internat. Congress Oto-Rhino-Laryngol.*, p. 104, 1953.

2. ZOLLNER, F.: Horverbessernde Operationen bei Chronischentzundlichen Prozessen Beider Mittelohren. *Ztschr. fur Laryngol., Rhinol. und Otol.*, 29:578, 1950.

3. LEMPERT, J.: Improvement of Hearing in Cases of Otosclerosis: A New One-Stage Surgical Technic. *Arch. Otolaryngol.*, 28:42, 1938.

4. ROSEN, S.: Mobilization of the Stapes to Restore Hearing in Otosclerosis. *N. Y. Jour. Med.*, 53:2650, 1953.

THE SURGICAL MANAGEMENT OF AGENESIS OF THE EXTERNAL AUDITORY CANAL BY MODIFICATION OF THE RAMBO MUSCULOPLASTY TECHNIQUE.*†

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The surgical management of congenital atresia of the external auditory canal is one of the most difficult problems confronting the aural surgeon. A basic knowledge of the embryologic development of the ear is essential for adequate management of these patients, and the interested reader is referred to the classical papers of Anson and Bast[1] and Altmann[2] on this subject.

The first operation appears to have been performed by Kiesselbach in 1883 on a child, aged six months, as reported by Fraser.[3] Dean and Gittens[4] reviewed operative techniques prior to 1917, crediting Schriebe in 1908 with opening the antrum from the mastoid and obtaining a wide canal by lining the bone with Thiersch grafts and at the same time reporting a case of their own similarly handled. In each case the incus was found to be mobile. In 1947 Pattee[5] reported five cases in which an endaural mastoidectomy was followed by primary skin grafting (Thiersch) to either the ossicles, if present, or directly to the stapes without fenestration of the horizontal canal. Ombredanne[6] favored immediate fenestration as a one-stage operation, Ruedi[7] advocated a two-stage procedure, and Shambaugh[8] reported 27 cases in which either a one-stage or a two-stage operation was performed.

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Early restoration of hearing is essential to the development of normal speech. Livingstone[9] prefers to operate before two years of age; Sullivan[10] at about four years; House[11] between this period and the start of formal schooling. The problems of postsurgical care in a very young child have led us to delay surgery until about six or seven years of age (First grade in school); therefore, we advise a bone conduction hearing aid at about two years of age to insure normal speech development in the interim.

Unilateral agenesis with normal hearing in the uninvolved ear is a contraindication to surgery for restoration of hearing. The advantage of binaurality seems minor in comparison to the rather formidable procedure required, and also there is the consideration that restored hearing is rarely normal and a reconstructed canal usually precludes swimming.

It is of the utmost importance to delay plastic surgery on the external ear until reconstruction of the external auditory canal has been completed, since frequently the ear remnant may be located well forward and inferior to the middle ear. It is relatively simple to plan an ear around an existing canal but almost impossible to move the avascular new ear, composed of skin and cartilage graft, any significant distance to oppose the ear canal and auricle. Five years and several plastic procedures were required to bridge a 3 cm. gap in one of our cases.

Congenital atresia combined with jaw and skull abnormalities (Treacher Collins syndrome) or mandibulofacial dysostosis presents the most trying problem. Anesthesia is hazardous due to the jaw deformity and concomitant laryngeal hypoplasia; mastoid cellular development is poor or absent, and the auricular deformities and displacement are frequently marked.

The use of full thickness skin grafts applied to bone and over ear cavities has not been completely satisfactory in our hands. Perforations of the graft, late necrosis, poor resistance to trauma and infection have been the principal problems. In an effort to obtain a more normal sized external meatus and at the same time provide for additional nourishment of the

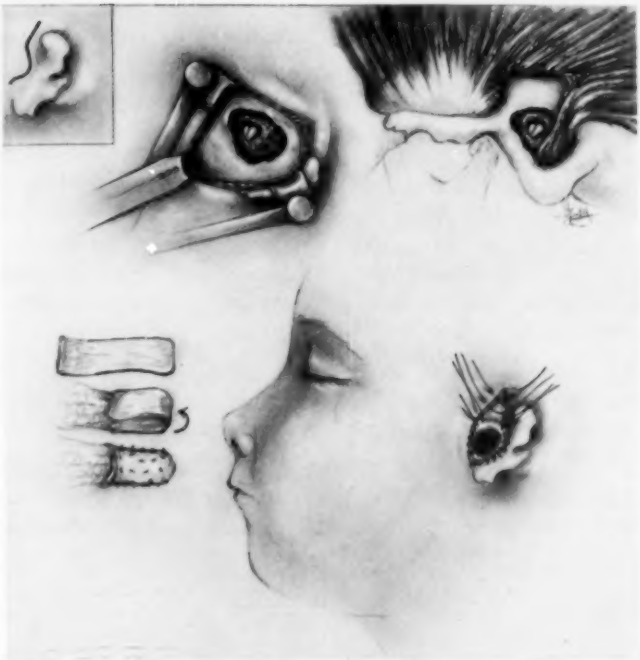

Fig. 1. The Use of Musculoplasty Flap in Reconstruction of the External Auditory Canal.

skin graft, a modification of the Rambo musculoplasty[12] technique was employed.

Three such procedures have been completed and found to be superior in all respects to the results obtained in seven similar cases on which more conventional techniques were used.

TECHNIQUE.

An incision resembling an inverted question mark is used so as to provide a tongue of skin for the "tragus" area of the future canal (see Fig. 1). A complete mastoidectomy is done by conventional techniques and the ossicular chain exposed.

The mobility and continuity of the ossicular chain, particularly the incus and stapes, are established by palpation.

A full thickness skin graft, .025 inches, is taken with the small dental engine-driven dermatome and formed into a sack over paraffin mesh gauze. The canal portions of the graft are lacerated for drainage, but the drum part is left intact. Livingstone has also described this very helpful method of applying the skin graft. The outer edges of the graft are trimmed and sutured to the skin incision with 5-0 chromic catgut which eliminates the painful process of removing these sutures later. The packing is removed on the tenth postoperative day.

The anterior flap of facial skin is undoubtedly a factor in preventing stricture of the newly formed canal as well as giving a more cosmetically acceptable appearance to the meatus.

In each of the three ears done with this technique the stapes was mobile and in contact with the incus. There was some deformity of the incus and malleus in each case, but this was of no functional significance.

RESULTS.

Age	Date of Surgery	Hearing	
		Preop. S.R.T.	Postop. S.R.T.
7	6-13-58	51 db	20 db
6½	6-29-59	45 db	22 db
7	4- 5-60	48 db	35 db

SUMMARY.

A modified technique for the restoration of hearing in congenital atresia of the external auditory canal has been presented. The use of the Rambo temporal muscle flap permits:

1. Complete mastoidectomy.
2. Small ear canal.
3. Well nourished graft.

Good hearing levels and less postoperative complications were

observed in the three ears done with this method when compared to seven ears done with more conventional procedures.

BIBLIOGRAPHY.

1. Bast, T. H., and Anson, B.: The Temporal Bone and the Ear. Charles C. Thomas, Springfield, 1949.

2. Altmann, F.: The Ear in Severe Malformation of the Head. *Arch. Otolaryngol.*, 66:7-25, July, 1957.

3. Fraser, J. S.: *Arch. Otolaryngol.*, 13:692-711, Jan., 1931.

4. Dean, W. W., and Gittens, T. R.: The Laryngoscope, 27:461-473, June, 1917.

5. Pattee, G. L.: *Arch. Otolaryngol.*, 45:568-580, May, 1947.

6. Ombredanne, P. M.: *Acta Otolaryngol.*, 41:69-109, 1922.

7. Ruedi, L.: The Laryngoscope, 66:148-151, Feb., 1956.

8. Shambaugh, G.: Surgery of the Ear. W. B. Saunders Company, Philadelphia, 1959.

9. Livingstone, G.: *Jour. Laryngol. and Otol.*, 72:9, 756-757, Sept., 1928.

10. Sullivan, J. A., McAskile, K., and Smith, B.: *Jour. Laryngol. and Otol.*, 73:4, 201-222, April, 1959.

11. House, H. P.: Management of Congenital Ear Canal Atresia. The Laryngoscope, 64:916, 1953.

12. Rambo, J. H. T.: Musculoplasty, A New Operation for Suppurative Middle Ear Deafness. *Trans. Amer. Acad. Ophthal. and Otolaryngol.*, 62:166-177, 1958.

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